THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—22ND YEAR. SYDNEY, SATURDAY, JULY 13, 1935. No. 2.

Table of Contents

An Address.[1]

THE HOSPITAL PROBLEM IN WESTERN AUSTRALIA.

By R. H. CRISP,

Retiring President, Western Australian Branch of the British Medical Association.

FOLLOWING the custom established by past presidents, I propose to address you briefly tonight, at the conclusion of my year of office, on a topical matter of medical politics. The subject of hospital policy is topical and vitally important to us as a profession. Indeed one might say that it is one of the most important problems before the Council. It is bristling with difficulties, and has been the subject of interminable discussions at the monthly meetings of your Hospital Policy Subcommittee.

[1] Read at the annual meeting of the Western Australian Branch of the British Medical Association on March 24, 1935.

We have progressed far from the original situation. Owing to the fact that our governments do not recognize liability to provide medical treatment for the sick poor, it was left to voluntary charity to build, equip and run a series of public hospitals. It was left to the medical profession to provide gratuitous treatment in these institutions for those who could not afford this privately. With expansions of these institutions, and with increase in complexity and cost of equipment and treatment, governments have had to assist financially to a greater and greater extent to keep the institutions solvent. Then there arose the practice of billing patients for the full cost of hospital treatment in order to try and swell the hospital revenue. This practice has seemed to give the public the right to attend the hospital on paying the costs.

The eligibility of patients for admission has been determined by an investigation of his financial standing as compared with certain standard income levels, these varying with the number of dependants.

But this inquiry has never been a strong department of the hospital, and there are many loopholes for abuse. Consider the hundred or two new patients coming up to the Perth Hospital any Monday morning and what time there is for the clerk to investigate each case on its merits. In some institutions it is no secret that, the more financial the patient, the more is he welcome as being able to pay his full hospital costs.

With increased efficiency, and not a little propaganda, patients who could pay are attracted to the hospital. Perhaps the hospitals mean well by their publicity and propaganda, hoping by this means to get donations and financial support, but on the whole its effect is pernicious. It seems to tell the public that the hospital provides treatment which cannot be obtained elsewhere. The Hospital Fund Tax in Western Australia would seem to lead taxpayers to expect some consideration in return for their contributions.

Financial depression has accentuated matters. Voluntary giving has dropped off, which has been another effect also of the Hospital Fund Tax. Also, the number of indigents has greatly increased.

The old-time spirit of independence and pride in paying one's way is passing. Increasing socialization provides more and more free bounties, such as free education, free kindergartens, free university, infant welfare clinics, protected industries, State subsidies, unemployment doles *et cetera*. Free medical treatment, which might be termed the "medical dole", is in danger of being considered a right if we continue to drift with the times.

The Federal Hospital Policy Subcommittee, with delegates from each State, came into being some two years ago, with the object of formulating a hospital policy acceptable to all States. This has involved the discussion by all States of the various drafts of the policy, and the suggested amendments and proposals emanating from these discussions. Ideas are gradually becoming crystallized and special difficulties becoming manifest, but there is still a great deal of work and thought necessary before anything can be finalized.

I should like briefly to touch on a few of the principal points emerging from this work.

Community hospitals are generally accepted in principle. All public hospitals should be changed over to community hospitals, allowing for the admission of intermediate and full-paying patients. We may say that in Western Australia we have government support for this principle. Most of our country hospitals are already community hospitals; a start has been made at the King Edward Memorial Hospital for Women, by the provision of a few intermediate beds, and there is a concrete proposal before us for changing over the Fremantle Hospital. The change over would automatically result in the disappearance of a great deal of hospital abuse.

The voluntary method of administration is preferred to State control, and there should be adequate medical representation on all hospital boards. We in this State have recommended to the Government the establishment of a hospital commission, on lines similar to those in New South Wales and Victoria. This commission would direct the change over to the community system, and would have wide advisory powers in correlating hospital work and development generally.

A community hospital should provide separate accommodation for paying and non-paying patients. This separation would of course not apply to special departments such as those of pathology, radiology *et cetera*. The "open system" should apply to the paying portion of the hospital, the patient being attended by his own doctor, even though the latter might not be a member of the honorary staff. The honorary staff would attend as at present to the non-paying department.

The definition of "ability to pay" is a major difficulty in formulating a policy. We believe that patients should pay "according to their means" for services rendered, including hospital maintenance, nursing and medical treatment. Discussion so far has not brought unanimity in defining those "unable to pay for medical treatment". Are we to adhere to the present system of free medical treatment for all those below a certain wage limit? This is the system that has led to so much abuse. It implies free medical treatment to the great majority of the wage-earning class. It has been said that four-fifths of the population would come into this category. At present a goodly proportion of these, the savers, manage to pay their way as private or intermediate patients, and have pride in doing so. They belong to lodges, they insure themselves, and they generally make provision for bad times by putting aside money for such contingencies. They are at present an important intermediate part of private practice. There is great danger that, under the old method of assessing eligibility by income limits, they would just naturally be drafted to the non-paying department of any community system.

Is it equitable that the hospital should collect the full hospital charge, say eight shillings per day or £2 16s. per week, before the doctor is able to make any charge for what is, after all, the main service rendered?

An alternative system of classification has been formulated and discussed at length by the Subcommittee of this Branch. Stated briefly, it is as follows:

The sick poor, or non-paying patients, would be defined as "those who are only able to pay contributions which, in their total, will amount to less than 10% of the combined cost of hospital accommodation *plus* medical treatment". They would be charged neither hospital nor medical fees. They would be considered a charge on the State, provision for which is made here by the Hospital Fund Tax. The classification would necessitate a rough estimate being made of the probable total cost of their stay in hospital, just as is done with private patients in deciding when they shall go. If the hospital stay becomes prolonged and complications ensue, a reclassification can easily be made.

Intermediate patients would be divided into two classes:

1. Low intermediate patients, certified by the inquiring authority as able to pay between 10% and 75% of their combined hospital and medical charges. The medical fees for these patients would be charged in accordance with an intermediate schedule, at about the same three-quarter level as our Workers' Compensation Schedule. The hospital would bill and collect all fees from this class, and all monies received, whether by lump sum or time payment, from a patient, would be entered in a ledger and credited on a *pro rata* basis to all concerned, namely hospital, doctor, consultant, radiologist, anæsthetist *et cetera*. Monthly or quarterly cheques would become available for those concerned. This would be a simple matter of bookkeeping.

2. Intermediate patients, those certified as able to pay 75% or more of combined fees. Medical fees for this class and for private patients would be billed and collected separately by the medical attendant concerned.

There would, no doubt, be some objections raised to such a system. Hospital boards would possibly consider they were being deprived of legitimate revenue; that they should be paid in full before any medical fee is collected. There would, however, be every incentive for the inquiring authority to place patients in their correct category, and there would be an end to the dunning by hospitals of the genuinely sick poor. Such a system would allow of the application of the principle, generally agreed to, that "patients should pay according to their means for hospital maintenance, nursing, and medical treatment".

The inquiring authority, whatever be the system of classification used, must however be greatly strengthened. This department of the hospital must contain experts. It is the keystone of the successful drafting of patients into their proper financial categories. Its efficiency is depended upon to protect from exploitation, not only the medical profession, but the hospital board, the Government, and so the taxpayer who finds the subsidy.

Health insurance is an essential part of any broad hospital policy. There have been hospital contributory schemes here in the past, such as that run by the Railways. These have, however, been mainly small, voluntary schemes sponsored by the hospital to cover public hospital charges.

In Sydney there is a big voluntary scheme, the Metropolitan Hospitals Contributory Fund, contributors to which are entitled, when ill, to a hospital benefit, available either for public or private hospital. There is, however, no medical benefit. A similar scheme has been mooted for this State, sponsored by the medical department.

Consensus of medical opinion is opposed to such schemes which do not include a medical benefit as well as a hospital benefit.

The Colonial Mutual Life Assurance Company tried an insurance, on the right lines, during the last year. It included a medical benefit. It had received the blessing of the British Medical Association in all States. It failed, but the experiment taught us some very valuable lessons. Even with the Colonial Mutual Assurance Company selling organization the policy was not taken up by the public in sufficient numbers to make it a paying concern. The reasons for this were probably first because it was voluntary, secondly because it covered illnesses treated in hospital only and not those treated at home, and thirdly because it was an individual, not a family insurance.

A further comprehensive voluntary scheme, with double benefits, is being launched in Melbourne, under the auspices of the Lord Mayor's Fund, the Victorian Branch of the British Medical Association, and other public bodies.

We must look in the future to some form of national health insurance to settle many of the remaining problems of hospital and national health policy. But any such insurance should be compulsory, at any rate for those whose normal financial position necessitates provision being made for the cost of an illness requiring hospital treatment.

It must include benefits making provision both for medical treatment and hospital costs.

There remain those on the basic wage, which does not include any provisions for illness, and those earning less than the basic wage. It might be claimed, justly I think, that the basic wage should include such provision for a health insurance premium. Some move for this adjustment may be necessary in the future. It is so much easier to drift into the present state of affairs than it is to bring matters back to a proper and equitable basis. Any suggestion, at present, of tampering with the basic wage would have tremendous repercussions on industry and on public opinion. We must perforce bide our time until a satisfactory health insurance scheme is a matter of practical politics. Those then would be regarded as the "sick poor", who could be defined as "those unable to contribute to health insurance". These, I think you will agree, would be "eligible for free medical treatment", and the profession would be proud to continue to treat them free, *gratis*, and for nothing. The cost of their hospital treatment could then well be considered a charge on the State. Pressure for medical fees in these cases could have but one result, a move towards nationalization of medicine.

REMARKS ON PYELITIS AND ITS MEDICAL TREATMENT.[1]

By A. R. SOUTHWOOD, M.D., M.S. (Adelaide), M.R.C.P. (London),
Honorary Physician, Adelaide Hospital.

Causes and Nature of Urinary Infection.

THE physician who has detected pus in his patient's urine finds himself engaged in fascinating problems of causation and treatment. The possible sources of pyuria are so diverse: the primary trouble may lie anywhere in the urinary tract or even in parts beyond. The treatment also presents most interesting features.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on April 24, 1935.

Some of the common causes of pyuria in the male subject I have set out in Figure I. In the female other possibilities exist; leucorrhœa is common, while rupture of a tubal abscess into the bladder is a rarer cause.

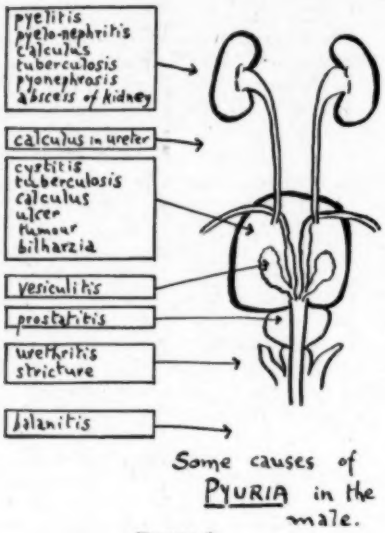


FIGURE I.
Some causes of pyuria.

It is at once obvious that not every patient whose urine contains pus is a sufferer from pyelitis. Indeed, it may well be asked, what is pyelitis? In its strict interpretation, the term implies an inflammatory condition limited to the upper expanded portion of the ureter. As such it probably never occurs. I am aware of no sound evidence that pyelitis unaccompanied by inflammation of the renal parenchyma ever exists. The transitional epithelium lining the ureter is carried up as a continuous sheet to line the renal pelvis. Figure II illustrates this direct continuity of lumen: from the myriads of Malpighian corpuscles in the kidney, through tubules, pyramids, papillæ and pelvis to the ureter, bladder and urethra. Once established in the urinary tract, infection has favourable conditions for its spread. The term "pyelitis" is too narrow; "pyelonephritis", heavy-sounding though it be, is the correct name for what we commonly call pyelitis. In this paper the terms are used synonymously.

General Lines of Investigation.

Several pertinent questions must be considered in every case of infection of the upper part of the urinary tract:

1. Where is the site of infection? In pyelitis the collecting tubules of the kidney and the lining of the pelvis are most involved.

2. What is the source of infection? Has it descended from the blood stream, through the kidney itself? Has it ascended from lower portions of the tract? Cohnheim in 1882 thought descending infection the commoner occurrence, but Winsbury White, in his Hunterian Lectures in 1933, considered that the almost invariable source was from parts below, the ascent being made via the periureteral lymphatics.

3. What is the bacterial cause? In about 85% of mild chronic cases of pyelitis *Bacillus coli* is to blame.

4. Is the trouble unilateral or bilateral? This can be accurately decided only by ureteral catheterization.

5. Are there any associated lesions? The important lesions are: (*a*) renal tuberculosis, (*b*) calculus, (*c*) neoplasm of urinary organs, (*d*) developmental abnormalities, (*e*) pressure from adjacent organs.

6. To what extent is the renal parenchyma damaged? Various tests of renal function may be applied to determine this.

7. Is there any obstruction present in the urinary tract? The history of the illness and the clinical examination may give some clues, but pyelography or ureteral catheterization are decisive methods.

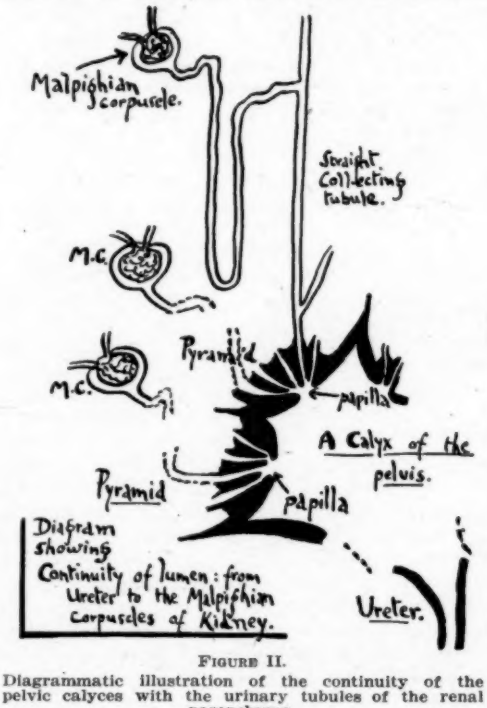


FIGURE II.
Diagrammatic illustration of the continuity of the pelvic calyces with the urinary tubules of the renal parenchyma.

Common Clinical Types.

Excluding cases associated with urinary calculi, tuberculous disease or neoplasm of the urinary tract, the remaining cases of pyelitis fall for the most part into a few groups. The following types are frequently met with:

1. Pyelitis of young infants, especially girls. Striking features are the sudden onset, often with a rigor, and the high and irregular pyrexia. The summer triad in infants—otitis, enteritis, pyelitis—is worth remembering.

2. Pyelitis in childhood. Girls are more often affected than boys. The onset is acute, with abdominal and lumbar pain and rigidity of the abdominal wall. The frequent resemblance of right-sided pyelitis to appendicitis is to be noted; in 15 of 100 cases of pyelitis among patients admitted to hospital the original diagnosis was appendicitis.

3. Pregnant women. The right kidney is the more frequently involved in this group. The anatomical relationships of the right ureter render it more exposed than the left to pressure at the pelvic brim.

4. Other adults, especially middle-aged women the subjects of leucorrhœa, and men with prostatic disease.

Management of the Acute Stage.

I use the term "stage" rather than "case", for there is almost always a chronic stage later to be dealt with. Microscopic examination of the urinary sediment is necessary at the outset, and progress is to be observed by weekly examination and culture. Rest in bed, ample bland fluids to drink, adequate aperients, and the application of heat to the painful side are the important points in treatment. Alkalis in large doses are to be given. Potassium citrate or bicarbonate or sodium bicarbonate may be given in four gramme (one drachm) doses every two hours at first, and later three or four times a day. The urine must be kept definitely alkaline to litmus. Alkalosis need not be feared in these cases, and up to 30 grammes (500 grains) of alkali *per diem* is a safe amount to give.

In the subacute stage, when painful symptoms have subsided, urinary antiseptics are useful. For many years efforts have been made to find some drug which, after ingestion or injection, is eliminated through the kidneys in sufficient concentration to give antiseptic properties to the urine. It is doubtful if any of the drugs to which powers in this direction are ascribed by manufacturers are really of much value. Recent work at Toronto University indicates that when hexamine, properly used, fails to clear a persisting urinary infection no other drug is likely to succeed. Hexamine is effective only if the urine is kept highly acid; it is necessary to stop the alkalis and to give some acid-producing substance. A mixture of hexamine, 0·6 gramme (ten grains), and ammonium chloride, 1·2 grammes (twenty grains), keeps well in solution for at least a fortnight and may be given four times a day. Hexamine acts best if the fluid intake is restricted. If dysuria recurs after taking hexamine for a few days, it is wise to revert to the alkaline method till comfort is regained, and then to go on with hexamine and ammonium chloride again.

Importance of the Reaction of the Urine.

The acidity of the urine is important in relation to bacterial growth in the urinary tract. In these days hydrogen ion concentration is much talked of, but a clear idea of the import of that high-sounding term and of its mystic symbol, pH, is not readily grasped by many of us. It is sufficient to our purpose if we remember that pH 7 indicates a neutral solution, while solutions having pH less than 7 are acid, more than 7 alkaline. The effect of the hydrogen ion concentration on the growth of *Bacillus coli* is indicated in Figure III. If the urine could be kept more alkaline than pH 9·5 or more acid than pH 5, *Bacillus coli* would cease to flourish in it. Such a degree of alkalinity cannot

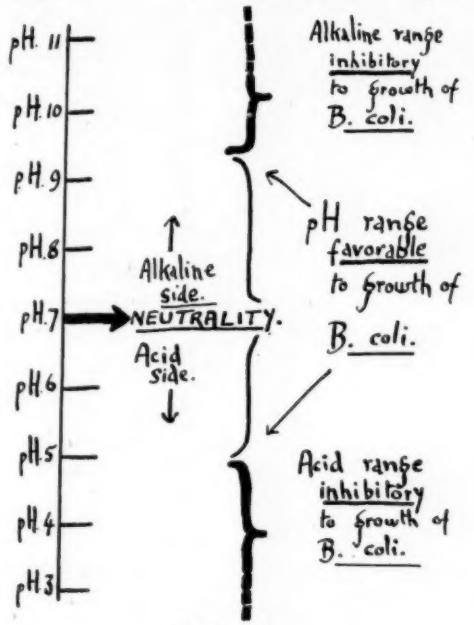


FIGURE III.

Hydrogen ion concentration scale, showing the reaction ranges favourable to *Bacillus coli* growth and the range inhibitory to its growth.

be maintained, but the acidity may be reached and kept by special methods. Numerous chemical indicators are useful in testing the pH of a solution. Figure IV shows three common indicators and their points of colour change. The application of these facts will be evident when we consider the use of ketogenic diets.

Ketogenic Diets in the Chronic Stage.

If pyuria persists in spite of treatment by alkalis and hexamine for four or five weeks, the next step is to put the patient on a ketogenic diet. This method is now the treatment *par excellence* for the chronic stage of pyelitis. The principle involved is the production of acidosis and associated ketonuria. At first glance this seems a very curious idea. For years physicians have laboured to combat acidosis in diabetes, in cyclical vomiting, and in post-anæsthetic vomiting, and now it is proposed deliberately to induce such a condition. We are setting a thief to catch a thief. We act in a similar strange manner when we infect general paralytics with malaria.

Ketogenic diets were first used in treating epilepsy, and it was observed that in these circumstances urine was slow to decompose. The experi-

mental work of Helmholz at the Mayo Clinic showed that this urine possessed antiseptic power.

When the fatty acid/glucose ratio of a diet exceeds 1·5, the subject is likely to show acetone and diacetic acid in the urine. On ordinary diets, such as those consisting of 300 grammes of carbohydrate, 100 grammes of protein, 100 grammes of fat, the ratio is only 0·4, and if the subject's metabolism is normal ketonuria does not occur. With the older types of diabetic diets we sailed much closer to the wind; 100 grammes of carbohydrate, 100

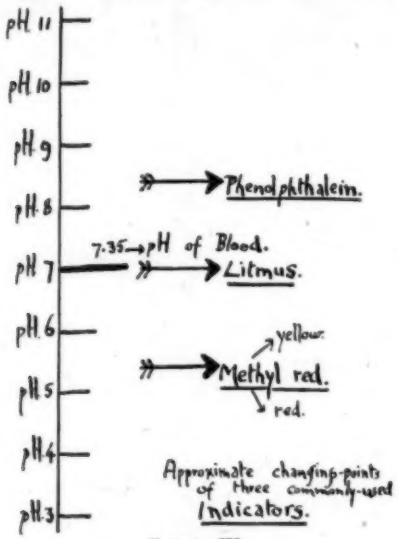


FIGURE IV.
Diagrammatic illustration of the use of indicators in reaction estimation.

grammes of protein, and 190 grammes of fat give a ratio of 1·3. A diet planned for the treatment of chronic urinary infection must show a still greater preponderance of fats; 20 grammes of carbohydrate, 70 grammes of protein, and 235 grammes of fat give a ratio of 2·9, and a heavy ketonuria is rapidly produced. This is an example of a suitable ketogenic diet:

Breakfast.

2 ounces bacon.
1 ounce butter.
1 ounce thick cream.
1 egg.
1 ounce milk.
2 ounces "5%" vegetables.
3 "Glutamon" or bran biscuits.

Dinner.

2 ounces meat.
1 ounce butter.
1 ounce cheese.
1 ounce thick cream.
2 ounces "10%" vegetables.
4 ounces "5%" vegetables.
3 "Glutamon" biscuits.

Tea.

2 ounces bacon.
1 ounce butter.
1 ounce thick cream.
1 egg.
1 ounce milk.
4 ounces "5%" vegetables.
3 "Glutamon" biscuits.

Carbohydate = 25, protein = 97, fat = 221. Calorie value = 2,500. Fatty acid/glucose ratio = 2·4.

Such diets are distasteful to most people; an iron will and a strong urge to be cured are essential to success.

Indications for Ketogenic Dieting.

Suitable conditions for this method of treatment are:

1. Subacute or recurrent cases of pyelonephritis or cystitis. Pregnancy pyelitis is unsuitable.
2. Chronic urinary infection without demonstrable gross lesions.
3. As a prelude to operation for underlying pathological condition in cases of urinary infection.
4. Post-operative urinary infection.
5. Urinary infection associated with inoperable neoplasm.
6. *Bacillus coli* infections are more responsive than those due to other organisms.
7. The patient should be robust enough to tolerate the diet, and the renal function should be of good to moderate grade.

Principles of the Diet Treatment.

The main points to be observed in carrying out treatment by means of the ketogenic diet are:

1. The diet is quantitative. The daily diet prescription varies with individuals. For the average adult 10 to 20 grammes of carbohydrate, 80 grammes of protein, 250 grammes of fat are suitable amounts. For a child of 25·2 kilograms (four stone) give 10 grammes of carbohydrate, 40 grammes of protein, 150 grammes of fat.
2. Give ammonium nitrate or chloride, 5·4 to 6·0 grammes (90 to 100 grains) *per diem* for three or four days before commencing the diet. This assists in gaining prompt and effective ketonuria.
3. Introduce the diet change abruptly; gradual training hinders ketosis. Ketonuria should develop in two days.
4. Do not continue diet beyond fourteen days.
5. Test each urine specimen with ferric chloride solution for ketones, and with methyl-red solution for reaction (pH 5·5).
6. Culture urine and stain sediment. Do this every three or four days. Four negative cultures are desirable. Figure V indicates the changes to be expected.
7. Absolutely strict adherence to diet is essential. Half measures are futile.
8. A moderately restricted fluid intake is desirable.

Special Investigations.

If after the maintenance of heavy ketonuria and high acidity for a fortnight the urine still contains bacilli, more searching investigations must be made. These are of three types:

1. Renal function tests. There are many to select from. I prefer the following: (*a*) water test of Volhard, (*b*) urea concentration test, (*c*) phenol-sulphonephthalein injection, (*d*) concentration of urea in blood, (*e*) urea clearance test.
2. Radiological investigations: (*a*) Plain X ray examination of the urinary tract. (*b*) Intravenous or excretory pyelography with "Uroselectan-B" or "Per-abrodil".

3. Urological examinations: (*a*) Cystoscopy and ureteral catheterization. Specimens of urine are obtained, representing the excretion of each kidney separately. In many cases the examination also

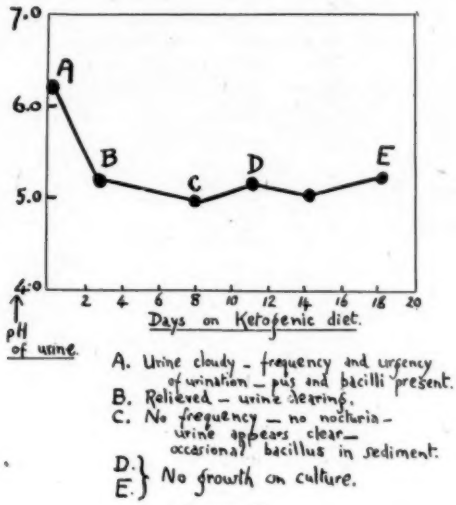


FIGURE V.

The effect of ketogenic diet on urinary reaction and on the signs of urinary tract infection. (After Cabot.)

serves as an excellent means of treatment by assuring drainage. Lavage of the renal pelvis may also be done. (*b*) Ascending pyelography.

It might be thought that pyelography would serve in diagnosing pyelitis, but there is no distinctive pyelogram in these cases. Slight blunting of the outline of the calyces is the likeliest evidence, but even this shows an early stage of complicating internal hydronephrosis. Figure VI illustrates the point.

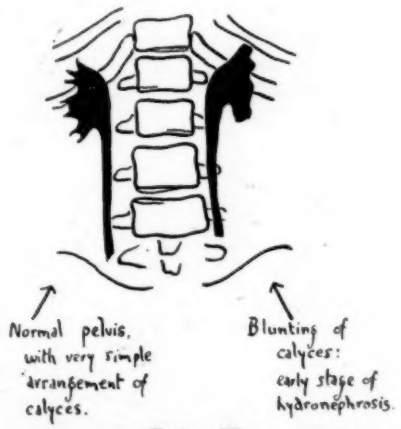
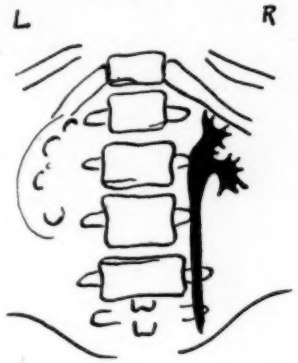


FIGURE VI.

Pyelogram demonstrating a normal pelvis and one in an early stage of hydronephrosis. (After Young.)

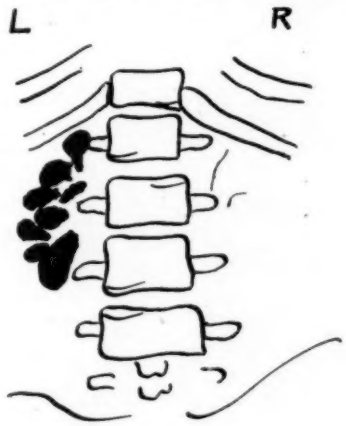
A girl of thirteen years who recently came under my care appeared to be suffering from simple acute pyelitis. Pyrexia and pain disappeared after a few days on alkalis, but pus cells and colon bacilli persisted in spite of medical measures. "Per-abrodil" pyelograms (Figures VII and VIII) showed the reason. In the affected kidney there was great delay in the elimination of the drug, and a severe degree of internal hydronephrosis was obvious. Nephrectomy is necessary.



Pyelogram obtained 10 minutes after intravenous injection of per-abrodil. (Patient L.P.).

FIGURE VII.

Pyelogram obtained ten minutes after intravenous injection of "Per-abrodil" (Patient L.P.), showing normal right pelvis and abnormal left pelvis. The elimination of "Per-abrodil" is much delayed on the left, and the outline is of advanced hydronephrosis.



Pyelogram obtained 40 minutes after intravenous injection of per-abrodil. (Patient L.P.).

FIGURE VIII.

Pyelogram obtained forty minutes after intravenous injection of "Per-abrodil" (Patient L.P., illustrated in Figure VII). The gross dilatation of calyces on the left side is obvious.

Summary of Treatment of Pyelitis.

This summary does not apply to patients in whom the history and clinical examination lead the physician to suspect calculus, tuberculosis or

neoplasm. When pyelitis seems uncomplicated by such gross lesions, the suitable plan is:

1. In the acute and subacute stages: bed, alkalis and, later, hexamine.
2. Resistant, recurrent and chronic cases when previous treatment has failed: ketogenic diet.
3. Still resistant infection demands the special investigations described.
4. Septic foci in urinary tract (prostate) or elsewhere (teeth, tonsils) are to be attended to.
5. Pregnancy pyelitis cases, unless responding to alkalis and hexamine, are best treated by ureteral catheterization.

My remarks have related mainly to the treatment of *Bacillus coli* infections by the use of the ketogenic diets. Of infections due to other organisms and of some other accepted methods of treatment I have said nothing. The use of vaccines, of *Bacillus acidophilus* treatment, and of high colonic lavage are all worth discussing.

Pyelitis unaccompanied by gross lesions is essentially a physician's problem, and these uncomplicated cases are suitably treated on the lines elaborated in this paper. When medical treatment, carefully applied for eight weeks or so, fails to cure the trouble, surgical procedures should be adopted. When pyuria is associated with an obstructive or destructive lesion in the urinary tract, indications for surgical attack may be obvious at the outset. In the absence of such indications the case is best left with the physician, for pyelitis will usually respond to vigorous, well-directed medical measures.

GASTRIC ULCER FROM THE SURGICAL POINT OF VIEW.[1]

By HOWARD BULLOCK, B.Sc. (Oxon), M.B., Ch.M. (Sydney), F.R.C.S. (England),

Honorary Surgeon, Sydney Hospital; Honorary Surgeon, Renwick Hospital for Infants, Sydney.

THE advent of McLean a little more than a decade ago, and the free use of alkalis, gave a tremendous impetus to the medical treatment of peptic ulcer; but fashions change, and with the deeper appreciation of the significance of chronicity in gastric ulcer an undoubted swing back to surgery has taken place.

It is not possible to do adequate justice to so vast a subject as "Gastric Ulcer from the Surgical Point of View" in one short hour, but I shall endeavour to put before you opinions and facts showing that surgery has a real and very important place in the treatment of chronic ulcer of the stomach.

You might ask: "Why not peptic ulcer?" "Why separate duodenal from gastric ulcer?" Moynihan[1] writes:

> There are gastric ulcers and there are duodenal ulcers. The difference between them is not merely geographical. These two forms of ulcer occur as a rule in opposite types of individuals, the pyloric sphincter forms between them a barrier very rarely overstepped, and finally a gastric ulcer becomes malignant in a formidable proportion of cases, whereas malignancy in a duodenal ulcer is an event so rare that few pathologists have seen it.

But as D. P. D. Wilkie[2] points out, gastric and duodenal ulcers often occur in the same individual; and in a series of 300 cases of ulcer of the stomach and duodenum he observed coincident duodenal and gastric ulceration in 42 patients, that is, in 14%. The surgeon, then, is confronted with the problem of adequate treatment of this double condition in the same patient.

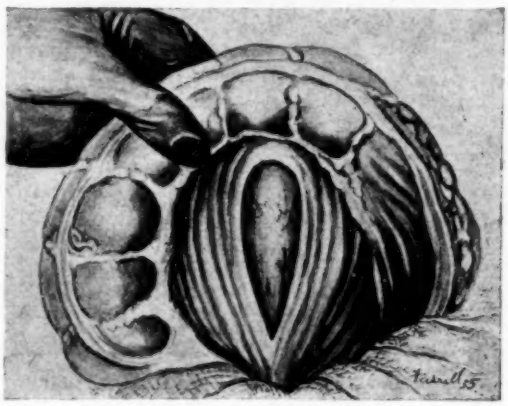


FIGURE I.

Posterior gastro-enterostomy. Transverse colon held up by hand. Transverse mesocolon slit, showing greater and lesser curvatures of the stomach.

FIGURE IIA shows position of opening in jejunum when clamps are used for posterior gastro-enterostomy.

FIGURE IIB shows resultant kinking of jejunum after using clamps for posterior gastro-enterostomy.

FIGURE IIC shows result when no clamps are used for posterior gastro-enterostomy. Note absence of kinking of jejunum.

The problem of gastric ulcer is a difficult one; medical and surgical treatment are interdependent, and the subject must be viewed from that standpoint.

[1] A post-graduate lecture delivered at Sydney Hospital, May, 1935.

I shall first of all put before you some slides to refresh your memories of earlier youth. The stomach is divided physiologically into a fundus, which harbours the oxyntic or acid-producing cells—

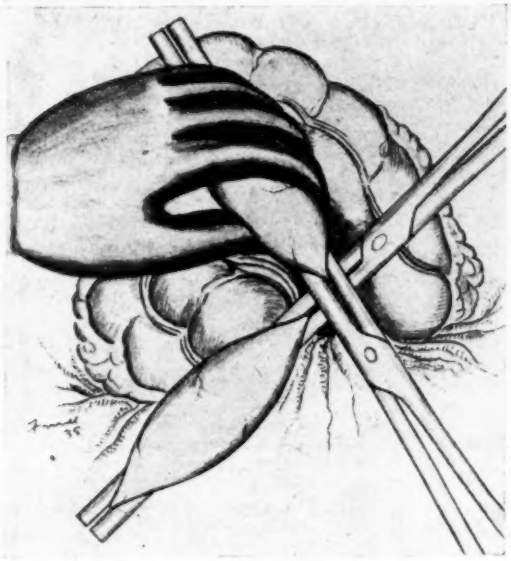


FIGURE III.
Posterior gastro-enterostomy. Shows the stomach through the transverse mesocolon, the lesser curvature being approximated to the proximal portion of the jejunum in the lower clamp. Note the trauma caused to achieve the above position of the gut.

a fact I wish you particularly to note—an antrum and a pyloric canal which is cut off from the duodenum by the pyloric sphincter muscle. The stomach is covered anteriorly and posteriorly with peri-

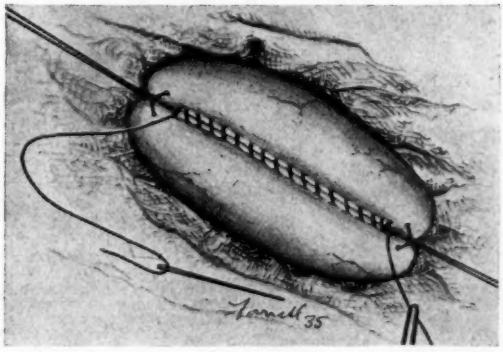


FIGURE IVA.
Posterior gastro-enterostomy. The stomach and jejunum approximated by guy sutures. Peritoneal suture in position.

toneum. In a normal stomach, when the percentage of hydrochloric acid of its contents rises to about 0·2%, the pylorus relaxes and allows regurgitation of the alkaline juices of the duodenum into the stomach. The stomach lies in close proximity to the liver, pancreas and colon, and it may involve each or all in its inflammations.

Ætiology.

The cause of chronic gastric ulcer is still debatable, but one fact is definite: it has not yet been proved that the chemical action of the gastric juice is capable of producing ulceration in an absolutely intact stomach mucous membrane.

Arthur F. Hurst[3] suggests that the types of stomach which predispose to the development of

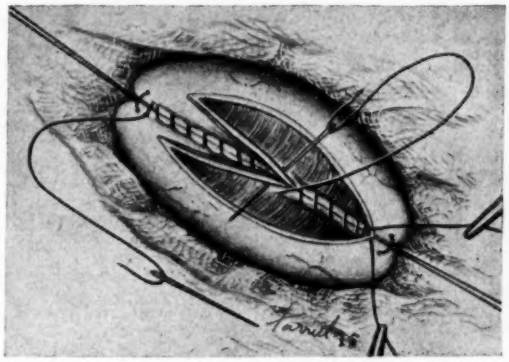


FIGURE IVB.
Posterior gastro-enterostomy. Peritoneal and muscular coats of stomach and jejunum being approximated. Mucosa exposed. Compare with Figure III for absence of trauma.

gastric and duodenal ulcer respectively are congenital and that either one or the other may exist in several members of a family; in fact, that there is a definite gastric ulcer and duodenal ulcer diathesis.

W. Langdon Brown,[4] in a learned article, "Predestination in Disease", pursues very much the same theme as Hurst.

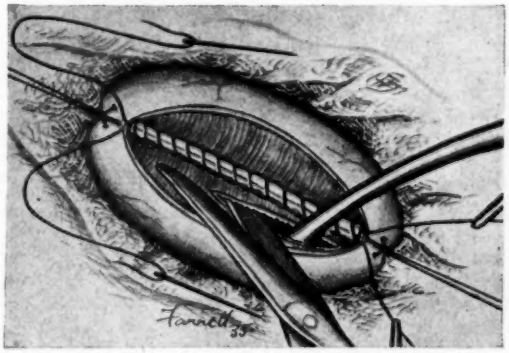


FIGURE VA.
Posterior gastro-enterostomy. Mucous membrane of jejunum being opened. Sucker in position removing digestive juices and blood.

E. C. Rosenow[5] produced ulcers in the stomach and duodenum of dogs by injections of streptococci isolated from the tonsil, but was unable to produce ulcer of the stomach by feeding with bacteria, except in starving animals, even when sharp particles were added to the food.

Berg,[6] with co-workers, deprived dogs of their pancreatic and biliary juices by means of fistulæ

and ligation of ducts. They conclude that the absence of alkaline secretions and their neutralizing effect on gastric acidity are not important in producing peptic ulceration, and, further, that gastric acidity is regulated by factors other than regurgitation of alkaline duodenal fluids.

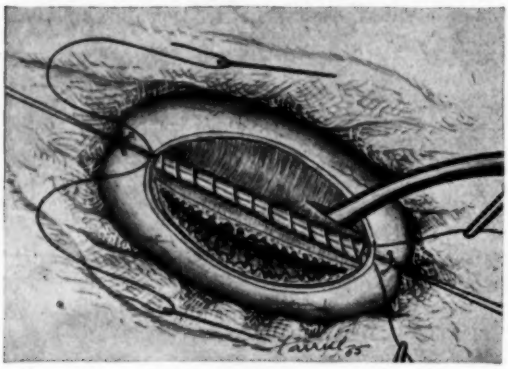


FIGURE Vb.
Posterior gastro-enterostomy. Cutting of jejunal mucosa completed. Stomach mucosa opened. Sucker removing any fluids within.

Miller[7] thinks that the starting point of gastric ulcer is in an infected lymphoid follicle.

Other factors suggested are injury caused by or associated with arterial spasm, hæmorrhage into local tissue or embolic infarcts, portal stasis with variations in the local blood pressure and consequent hæmorrhage with necrosis, chemical and thermal trauma.

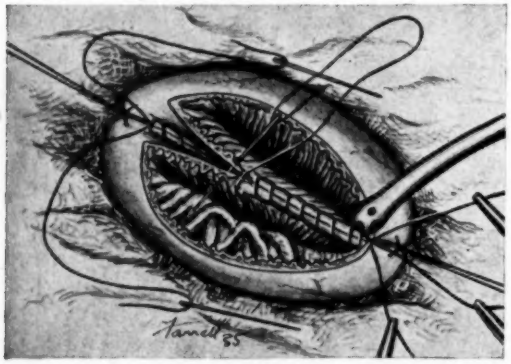


FIGURE VIa.
Posterior gastro-enterostomy. Mucous membrane of stomach and jejunum being approximated by third suture.

Harvey Cushing,[8] in his researches, found gastric, duodenal and even œsophageal ulceration in association with intracranial tumours, and is disposed to think that there may be a definite neurogenic basis of ulcer formation.

Piecing together the fragments, we may say that destruction of an area of stomach mucosa takes place and then, if the stomach is functioning unphysiologically, the acid completes the picture of erosion.

Symptoms and Signs.

A gastric ulcer may be a simple erosion and may be healed in three or four weeks by immediate and appropriate medical treatment if the health of the individual is good and the functions of the stomach are normal. If not taken immediately in hand,

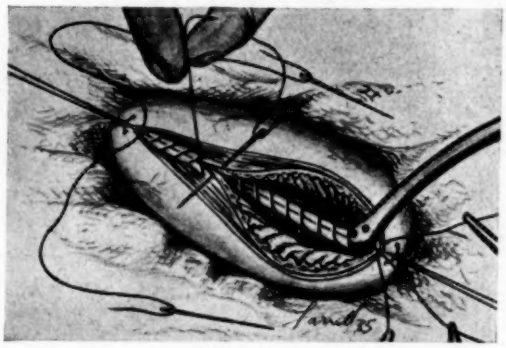


FIGURE VIb.
Posterior gastro-enterostomy. Third suture returning approximating outer mucosal edges.

chronicity is inevitable, the ulcer penetrates more deeply to the muscular wall and eventually erodes through the peritoneal coat to the free peritoneal cavity or into one of the surrounding organs, such as the liver, pancreas or colon. When it has reached this stage, the condition is definitely surgical and should be looked upon as such.

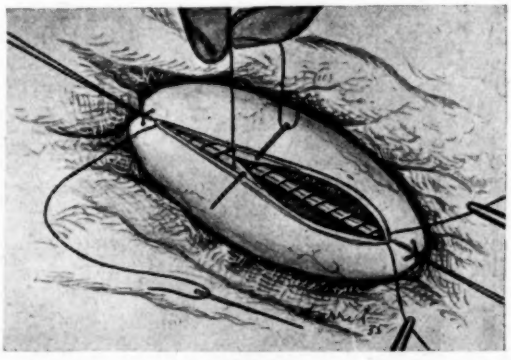


FIGURE VIIa.
Posterior gastro-enterostomy. The mucosal suture has been completed and the peritoneo-muscular suture (Number 2) is returning to the starting point.

An ulcer acts as an irritant affecting the neuromuscular mechanism of the stomach, the acidity of the gastric contents, and the amount of gastric juice secreted. The pyloric sphincter is specially affected, the output of stomach contents becomes irregular, the sphincter fails to relax, spasm and delayed emptying result. Regulation of the acidity of the gastric contents is interfered with, the percentage of hydrochloric acid in the stomach rises and maintains its height, even when the stomach empties, resulting in the condition known as hyper-

chlorhydria. Hypersecretion also supervenes from the prolongation of the digestive process.

The above applies mainly to ulcer near the pylorus. On the other hand, ulcer in the body of the stomach causes spasm at its location, but no blockage to the outflow of stomach contents unless the organ is of the hour-glass type, and leaves the contents of the stomach more or less normal.

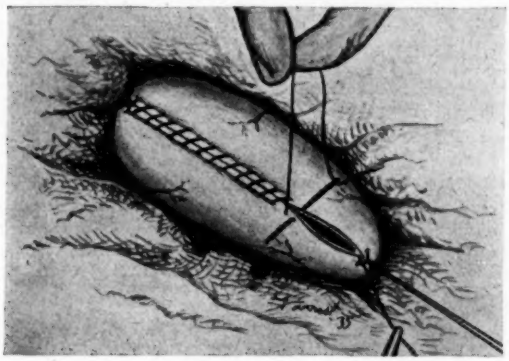


FIGURE VIIb.

Posterior gastro-enterostomy. The peritoneo-muscular suture has been completed and the peritoneal suture (Number 1) is returning to the starting point. Note that one guy suture has been cut.

Speaking generally and in a broad sense, the one constant symptom is pain in relation to meals. If the ulcer is close to the cardiac end pain will be felt immediately after meals and is progressively

FIGURE VIIIa.

Posterior gastro-enterostomy. Shows stomach stitched to opening in transverse mesocolon above anastomotic line.

later as the location of the ulcer approaches the pylorus, till at the pylorus pain may not be felt till two hours after meals. At the duodenum the pain is still later—up to three hours after food.

If erosion has taken place posteriorly or into the pancreas, pain may be felt in the back. Food generally relieves pain, as likewise do alkalis. Vomiting in gastric ulcer is quite common; hæmatemesis is rare. The symptoms are often brought on by indiscretions in diet, overwork, or worry or chills.

The fractional test meal is helpful in diagnosis, but not always, as the case of A.K., aged forty-seven years, shows.

The test meal was normal in every way, though X ray examination disclosed an ulcer in the *antrum pylori*. A carcinoma was removed last February by partial gastrectomy and entero-enterostomy, after the method to be described later. Since operation he has gained over 12·6 kilograms (two stone) in weight.

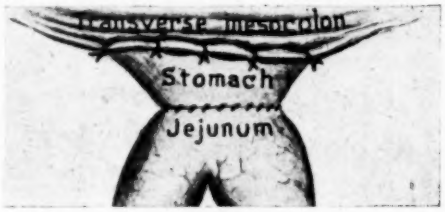


FIGURE VIIIb.

Posterior gastro-enterostomy. Shows result with definite guarantee against any kinking of the jejunum.

Examination for occult blood should be made, and care should be taken that the patient at the time is on a meat-free diet. Abdominal inspection and palpation are most important, and a large inactive ulcer may be present and no tenderness may be elicited on palpation. The final arbiter will be X ray examination, yet I shall now show you a case in which even five attempts by three different expert and reliable men failed to disclose ulcer.

The patient, R.M., aged forty-three years, as you will see, now enjoys the best of health and has gained over 12·6 kilograms (two stone) in weight since the operation, and eats anything. The operation was similar to that just mentioned above and was performed two years ago.

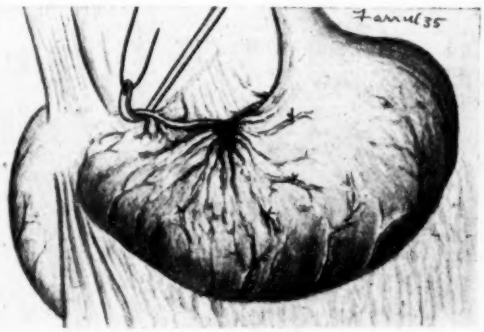


FIGURE IXa.

Resection of ulcer. Shows ulcer of lesser curvature of stomach with aneurysm needle under right gastric artery, with larger vessels entering the ulcer undermined and ligatured.

So there is the occasional case in which all help from the physical and chemical laboratory tests breaks down and we must depend on clinical evidence for diagnosis.

I have spoken of chronic ulcer—a condition that one can and should sit down and think over before

deciding on the lines of treatment—but acute perforation into the peritoneal cavity, to the greater or lesser peritoneal sac, demands active and urgent surgery.

Briefly, there is an acute onset of agonizing pain, board-like rigidity of the abdominal muscles preventing any examination of the subjacent organs. The patient is anxious of his condition and demands relief. There is generally a pulse of good volume, a

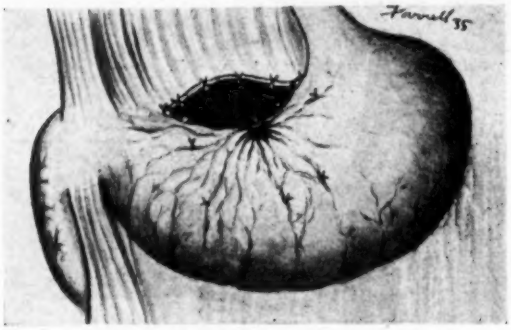


FIGURE IXB.
Resection of ulcer. Shows ulcer isolated from gastro-hepatic omentum, vessels entering the area tied off.

normal or slightly subnormal temperature, some nausea and vomiting. The breathing is rapid, shallow, jerky and thoracic. The abdomen is hyperalgesic; sweating of head and chest and shoulder pain are present. Later, pneumoperitoneum and loss of liver dulness appear. When free fluid can be detected there is generally abdominal distension, with cold extremities, subnormal temperature and rapid pulse; in fact, the signs of impending dissolution.

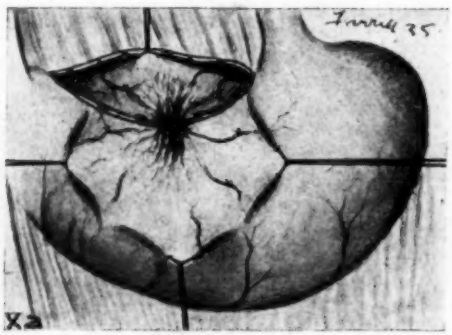


FIGURE XA.
Resection of ulcer. Guy sutures in position, with posterior surface of stomach brought into view.

Differential Diagnosis.

The chief difficulty in differential diagnosis with perforations will be pancreatitis, appendicitis, acute cholecystitis (particularly with rupture) and leaking Fallopian tubes.

A careful history will always help. In a series of thirty-two cases of rupture published by me,[9] a history of indigestion was found in every case, and many patients had submitted to a course of medical treatment.

The one outstanding sign is the board-like rigidity of the abdominal muscles, which do not relax for an instant. I have never seen it in any other condition. Temperature raised above a degree Fahrenheit at the most is never met with in early cases, nor is there any leucocytosis.

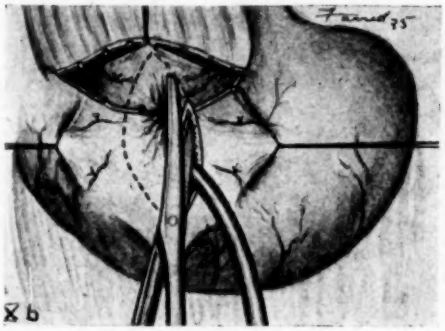


FIGURE XB.
Resection of ulcer. Sucker in position. Scissors cutting out ulcer.

One has to remember that acute perforation of cancer of the stomach does occur and may simulate ulcer. Ian Aird[10] discusses perforations in 71 cases, and in old people particularly this catastrophe must be kept in mind.

Treatment.

No surgeon who has had experience in gastric surgery could look upon the treatment of a gastric ulcer other than essentially within the realm of the physician, and upon surgery only as a last resort.

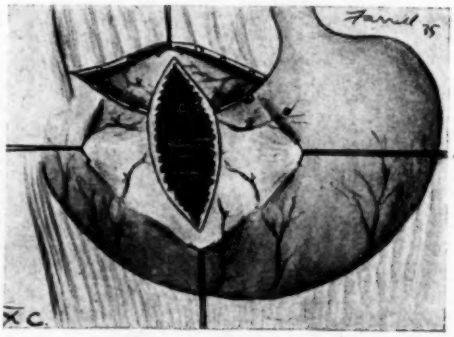


FIGURE XC.
Resection of ulcer. Ulcer removed. Stomach open for inspection.

We have noted with the presence of ulcer irritability of the stomach neuro-muscular mechanism and excess of gastric secretion and the tendency to hyperacidity.

Medical Treatment.

Medical treatment, then, should aim at lessening the neuro-muscular irritability and at reducing the amount of gastric secretion. As a preliminary, the patient should be at least six weeks in bed, with

dieting and drugs; the latter, of course, must be continued with ambulatory treatment.

As for diet, remember that meat extractives stimulate gastric flow and are to be avoided. Beef and mutton are more difficult to disintegrate than fish and poultry, stay longer in the stomach and excite the flow of gastric juice. Milk causes little flow. Raw egg has a minimum effect, while fats inhibit gastric secretion. Drugs should consist of alkalis and atropine.

The patient's resistance to infection must be raised and elimination of sources of infective absorption carried out. Abnormality of gastric juice should be corrected.

Alkaline treatment is not without danger, producing a condition of alkalosis, as W. F. Gatewood, O. H. Gaebler, E. Muntwyler and V. C. Myers[11] have pointed out. The alkalosis manifests itself with headache, nervousness, nausea, vomiting, pains in joints and muscles, weakness, slow respiration, rapid pulse, flushing of face and sweating.

With all this treatment and under the strictest medical supervision, there is a certain percentage of patients who will not respond and many more who will not persist in or cannot afford to carry out the dietary and lines of conduct of life laid down by their medical adviser. The ulcer progresses and tends to greater chronicity, causing further disordered stomach functions, contractures and deformity of the stomach, adhesions and penetration into the surrounding organs; the pancreas is peculiarly liable to invasion.

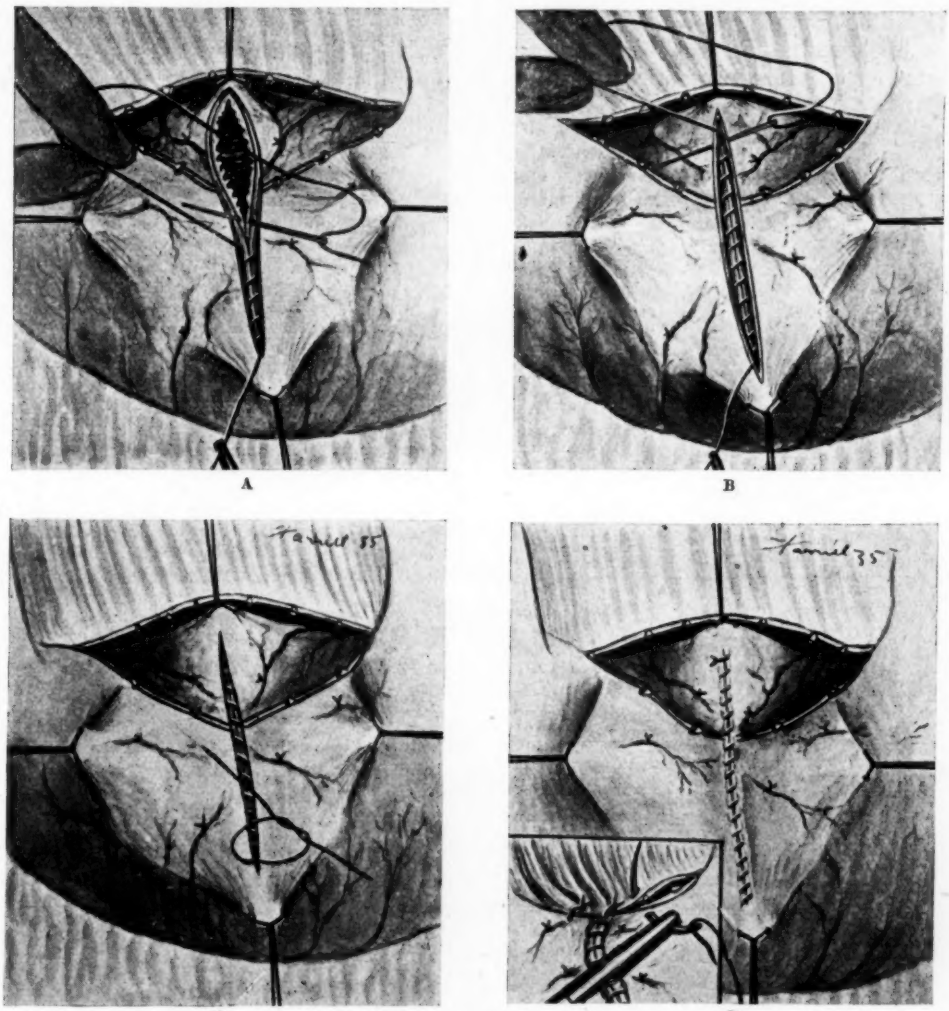


FIGURE XI. Resection of ulcer.

A: Mucous membrane being sutured. B: Same suture returning approximating muscular coats. C: Shows previous suture being tied. D: Peritoneal suture completed. Insert: Approximation of cut edges of lesser hepatic omentum.

That ulcers heal even after reaching large dimensions is unquestionable, for often large, sound scars are found in stomachs at operation, and in the *post mortem* room. Moynihan[1] quotes Stewart, who finds open or healed ulcers in 5% of the bodies examined at Leeds.

Now let me overwhelm you with some figures—statistics which can be illuminating, but equally, too, misleading.

Bolton[12] quotes Neilsen, who states that 88% of relapses occur in the first two and a half years, and if symptoms are present for more than five years, the probability of permanent cure is only 10%.

Conybeare[13] quotes Einhorn's and Crohn's and Hunter's and Balfour's figures, which make interesting reading, and which show the danger of chronicity in ulcer and the definite relationship between gastric ulcer and gastric cancer.

Winhorn and Crohn discuss 100 cases of peptic ulcer from 1922 to 1925 (four years). The figures quoted by J. J. Conybeare[13] are set out in Table I.

TABLE III.

Actual Deaths among 1,651 Patients Operated Upon for Duodenal Ulcer.		Expected Deaths among 1,651 of the General Population comprising Individuals of same Age Groups.	
Year Following Operation.	Number of Deaths.	Year.	Percentage.
First	22	First	24·0
Second	20	Second	18·6
Third	9	Third	14·3
Fourth	10	Fourth	11·2
Fifth	11	Fifth	8·3
Sixth	13	Sixth	16·7

TABLE I.

Remote Results of Medical Treatment in Percentages.

Year.	Number of Patients.	Period of Follow-Up in Years.	Cured or Improved.	Unimproved.	Operated Upon.
1922	22	4	50%	9%	32%
1923	17	3	65%	6%	23%
1924	30	2	67%	7%	23%
1925	32	1	89%	9%	3%

Thus the longer the period of observation after medical treatment, the greater becomes the proportion of relapses and the smaller the proportion of cures. Also, at the end of four years from the beginning of medical treatment, only 50% of the patients remain cured or even improved, and practically one-third of the patients had to submit to surgical operation.

Investigations of Balfour and Hunter, of the Mayo Clinic, 1906-1915 ("Collected Papers of the Mayo Clinic", Volume XVI, 1924, page 60) are quoted by Conybeare[13] (Tables II and III).

TABLE II.

Actual Deaths among 521 Patients Operated Upon for Gastric Ulcer.		Expected Deaths among 521 of the General Population comprising Individuals of same Age Groups.	
Year Following Operation.	Number of Deaths.	Year.	Percentage.
First	36	First	8·2
Second	21	Second	6·5
Third	11	Third	4·9
Fourth	7	Fourth	4·1
Fifth	7	Fifth	3·0
Sixth	6	Sixth	5·6

Note.—Mortality in the first two years following operation was much higher than the expected mortality among the control group. After two years the mortality gradually falls to approximately normal. The explanation of the high mortality in the early years lies in a mistaken diagnosis of gastric ulcer in patients really suffering from carcinoma.

Theoretically, therefore, patients who have been operated upon for duodenal ulcer are better lives than those who have never had an ulcer.

Moynihan[15] gives his twelve years' observations with Professor M. J. Stewart showing the incidence of chronicity with perforations and hæmorrhage as follows:

Of 61 patients with gastric ulcer dying from perforation, 60 had a chronic ulcer.

Of 14 patients with gastric ulcer dying from hæmorrhage, 13 had a chronic ulcer.

Of 117 patients with gastric ulcer dying from hæmorrhage, 109 had a chronic ulcer.

Of 12 patients with duodenal ulcer dying from hæmorrhage, all had a chronic ulcer.

A. M. Cooke[16] quotes the figures at Saint Thomas's Hospital from 1921 to 1931, inclusive, where there were admitted 2,517 patients with peptic ulcer and the general death rate was 8·9%. There were 1,198 operations with a mortality of 13·2%, whereas medical treatment was given to 1,319 with a mortality rate of 5%. But by excluding cases of late perforation with general peritonitis he gives the surgical mortality at 6%—1% higher than the medical treatment rate.

Does a gastric ulcer become malignant? I shall quote but a few authors on this question.

First, A. F. Hurst,[17] in the Schorstein Lecture, "The Precursors of Cancer of the Stomach",

expresses his opinion that 4·5% of cancers of the stomach are secondary to ulcer of the stomach and quotes Orator, whose figures are: of 34 prepyloric ulcers removed, 11 showed a malignant degeneration.

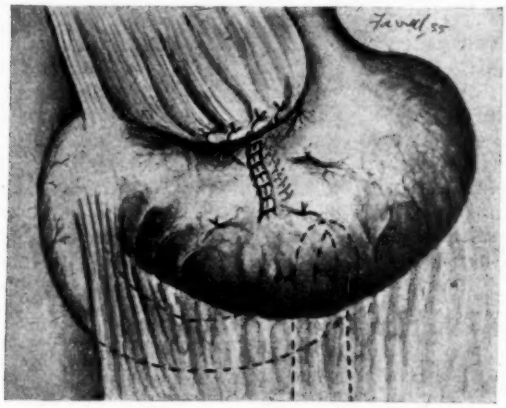


FIGURE XII.

Resection of ulcer. Operation completed, with posterior gastro-enterostomy. Note that artificial stoma is proximal to operation area.

A. J. Walton[18] considers 10% as a low estimate of cancer following ulcer.

Victor Pauchet[19] has observed 2,200 cases of cancer of the stomach in thirty years. He states

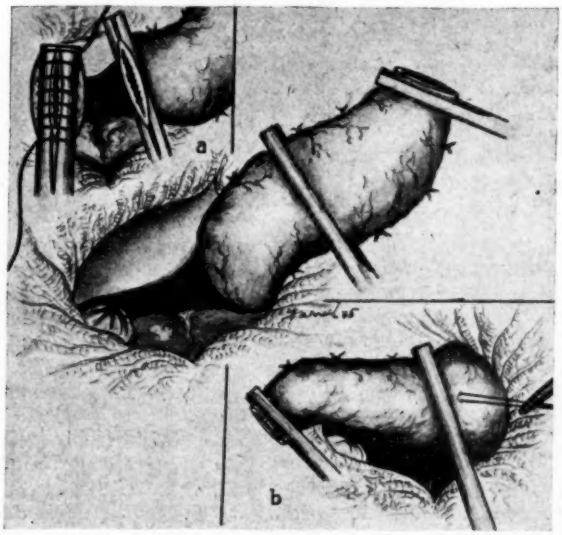


FIGURE XIII.

Partial gastrectomy. Stomach and portion of the duodenum have been freed and distal part of duodenum tied off. Insert a: Method of oversewing duodenum. Insert b: Guy suture in position on anterior surface of stomach, above line of excision. (Compare with Figure XVII.)

that 80% are grafted on chronic ulcer, many of which have remained simple for fifteen years. He further states that 50% of cancers of the stomach examined are incurable. His mortality rate for gastrectomy for cancer is 20%; his mortality rate for gastrectomy for ulcer is 2%.

W. L. A. Wellbrock,[20] pathologist at the Mayo Clinic, from examination of fresh frozen sections

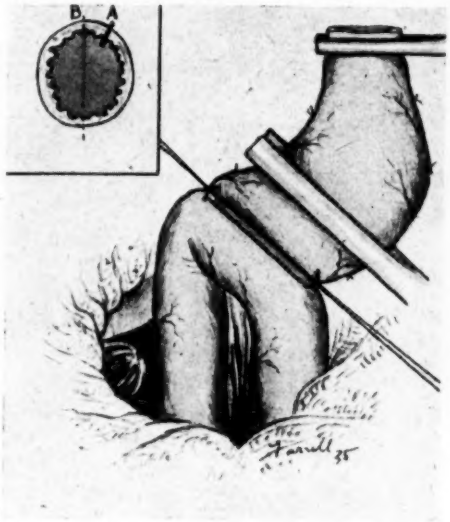


FIGURE XIVA.

Partial gastrectomy. Jejunum and stomach approximated. Guy sutures in position. Insert: Section of jejunum. A = attachment of jejunum to stomach. Dotted line B = opening of stoma.

of gastric ulcers excised came to the following conclusions. The differential diagnosis of benign and malignant gastric ulcers is notoriously defective.

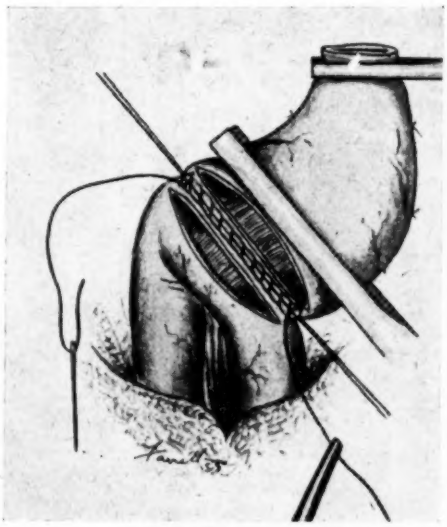


FIGURE XIVB.

Partial gastrectomy. Peritoneal suture (Number 1) inserted. Mucosa of stomach and jejunum exposed.

All chronic gastric ulcers are suspected of being carcinomatous and should be treated as such before and at the time of the operation. The use of the

microscope is the only means of distinguishing simple chronic ulcer from early gastric cancer. Diagnosis cannot be made by clinical means or Röntgenoscopy or by the appearance of the gross specimen.

FIGURE XVA.
Partial gastrectomy. Suture (Number 2) in position.

M. J. Stewart[21] observed 259 operation specimens. There were 70 cases of cancer, 11 arising in ulcer, that is, 15%. There were 180 cases of chronic ulcer, 11 with cancer, that is, 6·1%.

FIGURE XVB.
Partial gastrectomy. Jejunum being opened. Sucker being inserted.

W. J. M. Scott[22] discusses the relationship of carcinoma with callous gastric ulcer. His observations show that 15% to 20% of clinically diagnosed gastric ulcers become malignant.

Now let us consider the statistics of hæmorrhage from gastric ulcer treated medically.

J. J. Conybeare[13] reports that at Guy's Hospital from 1911 to 1920, 600 patients suffered from severe gastric and duodenal hæmorrhage. The mortality rate was 2%.

A. M. Cooke[16] reports that at Saint Thomas's Hospital from 1921 to 1931, 191 patients were admitted requiring urgent attention from sudden loss of blood by vomiting or by bowel. This total does not include patients in whom the serious loss of blood was not the predominating symptom. The mortality rate was 24·1%.

Gordon-Taylor[23] records that at the Middlesex Hospital from 1924 to 1933, 124 patients were admitted with hæmatemesis. Twenty patients died; the mortality rate was 24%. Ulcer was proven in 67. *Post mortem* examination showed that out of 20 who died, 16 had been suitable for surgery.

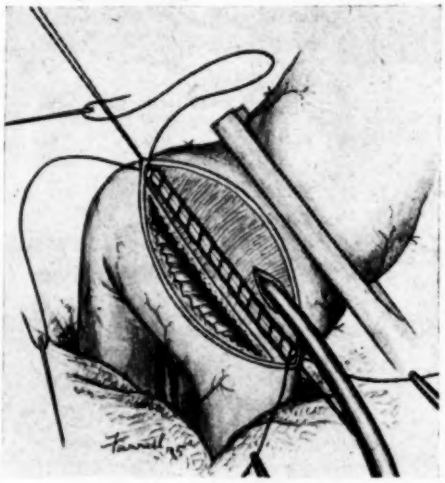


FIGURE XVIA.
Partial gastrectomy. Jejunum opened. Sucker in stomach

Keith Ross[24] records that at the Royal Melbourne Hospital from 1919 to 1928, 187 patients were admitted with hæmorrhage; 28 died. The mortality rate was 6·5%.

Stewart[25] records 9,000 consecutive autopsies. The mortality rate from severe gastric hæmorrhage was 25%.

And now for a short review of perforations.

John Watson[26] reports that in Great Britain twelve surgeons from 1896 to 1930 had 1,363 cases in which perforation occurred; 417 patients died. The mortality rate was 31·6%.

Moynihan[27] from 1920 to 1925 had 237 patients with perforation; 56 died. The mortality rate was 25%.

H. Searby,[28] writing from the Royal Melbourne Hospital, records 113 consecutive cases. The mortality rate was 30%. Among 33 personal cases, two patients died, a mortality rate of 6%.

H. Bullock[7] recorded 32 cases. Two patients died, a mortality rate of 6%.

Surgical Treatment.

Up to the present I have endeavoured to keep surgical treatment in the background and to present to you fairly figures from eminent physicians and pathologists, mostly British, showing that medical treatment of gastric ulcer has a definite death rate; that, in spite of what is generally accepted in this city, there is a death rate, and quite a high one, from hæmorrhaging ulcer; and that there is quite a high percentage of failures in medical treatment. And, moreover, most of the patients who come to the surgeon for perforation have submitted to medical treatment. Surely it is quite fair to lay at the door of the physicians some responsibility for failure to effect a cure, and quite fair that they should share in responsibility for the death rate.

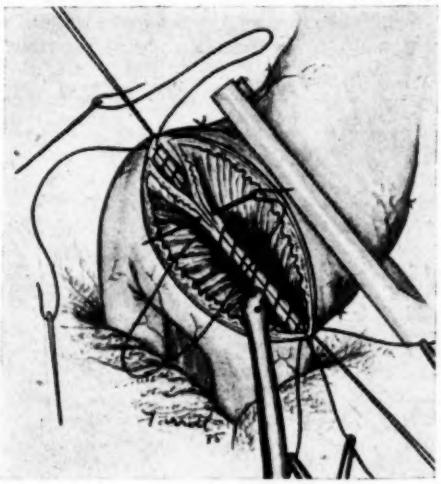


FIGURE XVIB.
Partial gastrectomy. Mucosal suture (Number 3) being inserted.

We have shown you that with ulcer of the stomach there is a general rebellion, pyloric spasm, hypersecretion, delayed emptying and consequently hyperacidity.

The aim of surgery will be to correct this by: (i) facilitating the emptying of the stomach; (ii) inducing regurgitation of intestinal fluids into the stomach, thereby neutralizing its contents in imitation of the normal duodenal regurgitation; (iii) removal of the lesion.

What, then, are the indications for surgery? Hurst[3] suggests the following: (i) perforation, (ii) pyloric obstruction, (iii) hour-glass stomach, (iv) no response to treatment, (v) one severe hæmorrhage, (vi) persistent occult blood and pain in spite of treatment.

Besides Hurst's suggestion, I shall put Balfour's views:[29]

If a gastric ulcer fails to heal, it should be classed as a lesion other than an ulcer. Even though symptoms are more or less quiescent, a well planned and well performed surgical operation is safer for the patient than harbouring a lesion which persists.

In 1927 I made an extended tour of America, Canada, and most European centres of note, and of Great Britain, and spent a very pleasant and instructive month in Vienna. The important surgical clinic of von Eiselberg was then being conducted by an extremely able surgeon, Schönbauer,

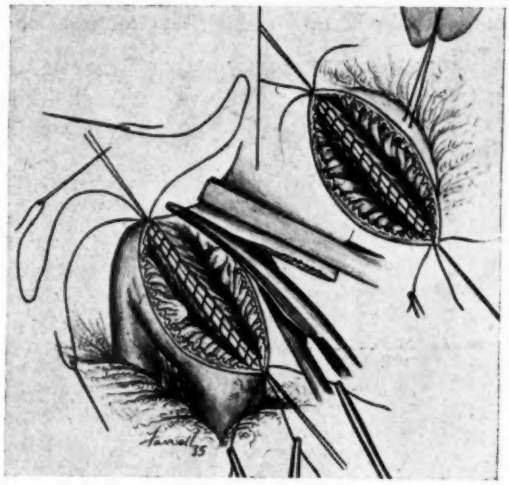


FIGURE XVII.
Partial gastrectomy. Stomach being freed. Insert: Guy suture referred to in Figure XIII.

who was removing ulcers by the dozen. His jargon was that typical of all of us. So I decided to see Ortner,[30] a physician who before the war was specially invited to spend a year giving instruction at Harvard, and whose book on abdominal pain is

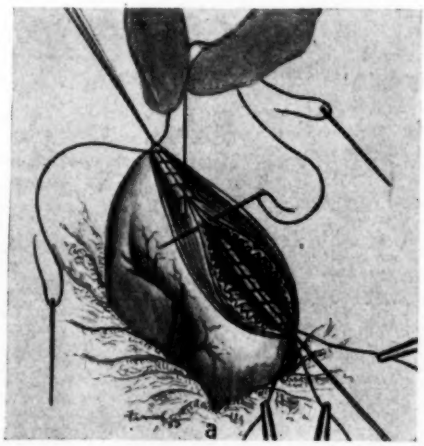


FIGURE XVIIIA.
Partial gastrectomy. Mucosal suture (Number 3) returning.

very readable and instructive. He disagreed with mutilating surgery, thought that the occurrence of cancer on ulcer was just a bogy (2% was a high estimate), took me to a window and showed me old women and men (fully a dozen) with ulcers,

basking in the sun and quite happy. He put it to me: "Why rob them of life by turning them over to the surgeon?" A Viennese viewpoint!

I have endeavoured to impress upon you that there is a definite association between ulcer of the stomach and carcinoma of the stomach. (*En passant* it is interesting to note that Hurst's[31] view is that in ulcer-cancer of the stomach the test meal shows

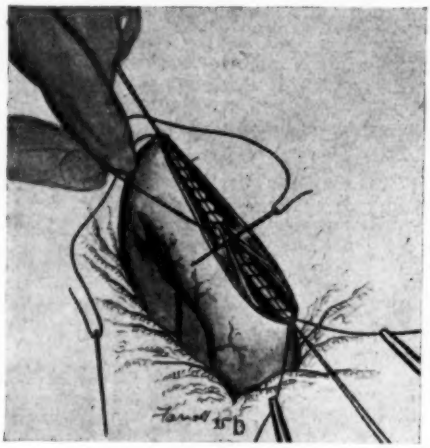


FIGURE XVIIIB.
Partial gastrectomy. Mucosal suture completed, muscular suture (Number 2) returning.

acidity, while in ulcers from cancer *ab initio* achlorhydria is always present.) Knowing this, then, must we not regard as within the province of the surgeon penetrating ulcers and ulcers that will not respond to treatment and that tend to relapse?

FIGURE XVIIIC.
Partial gastrectomy. Peritoneal suture completed. Guy sutures cut.

Many operations are practised for the relief of gastric ulcer, and many fancy procedures are suggested, such as vagotomy, adrenal denervation, partial devascularization, radiotherapy, physiotherapy, psychotherapy, protein shock *et cetera*.

Some years ago a distinguished surgical colleague informed me that he could recite 225 different ways of performing gastro-enterostomy. Since then I am sure many more have been invented, which indicates failure or, more correctly, a high percentage of failures from surgical treatment. Such a case I present to you.

E.H., aged twenty-eight years, had pyloroplasty performed four years ago by a capable surgeon. There was temporary relief. X ray examination showed delayed emptying of the stomach just before last February, when partial gastrectomy was carried out with entero-enterostomy. He has since gained over 12·6 kilograms (two stone) in weight and enjoys perfect digestion.

Connell[32] advocates fundesectomy to remove the cause (acid) instead of the effect (ulcer), the latter leaving the cause to remain. (We know, though, that acid alone is not the cause of chronic ulcer.)

The technical aim of the surgeon should be to carry out some procedure, simple as possible, with

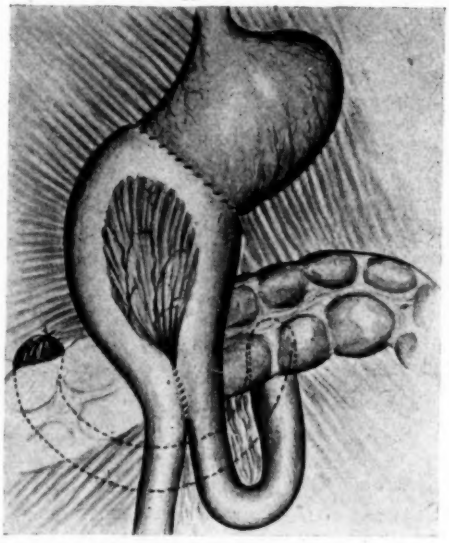


FIGURE XIX.
Partial gastrectomy. Completed gastro-jejunostomy with jejuno-jejunostomy. Note long jejunal loop.

the maximum of good effect to the patient; and partial gastrectomy in some form conforms to this standard, the pylorus being removed at the same time. In removing the pylorus, whether for duodenal or gastric ulcer, it appears to me that the hormonic balance between the digestive secretions is upset, with advantage to the patient, in lowering the acid secretion of the stomach. At the same time the stomach neuro-muscular mechanism, which we have shown above is at fault in gastric ulcer, is robbed of its irritability.

I feel sure that the power the pylorus has to cause gastric disturbance is not fully appreciated, and one has only to operate upon cases of congenital hypertrophy of the pylorus (and I have done a great number at the Renwick Hospital for Infants) to observe what a dramatic change is produced by rendering innocuous the pyloric sphincter.

Balfour's[29] mortality rate for this procedure, partial gastrectomy, is between 3% and 4%, and he points out that, fortunately, gastro-jejunal ulcer is rare after gastro-enterostomy for gastric ulcer.

Moynihan in all his writings admits about the same mortality rate. L. N. Broster[33] gives the Charing Cross Hospital figures for mortality rate at between 6% and 7%. J. J. Conybeare[13] gives the Guy's Hospital mortality rate at 5%. A. M. Cooke[16] gives the Saint Thomas's Hospital mortality rate at 6%.

There is, then, a definite mortality rate in the best of surgical hands, but the mortality rate from medical treatment before mentioned is approximately the same.

But surgery offers this advantage: removal of a focus potentially malignant, liable to perforate and certainly to hæmorrhage. With regard to the latter (hæmorrhage), Conybeare states that one in 400 patients with chronic ulcer dies from hæmorrhage, and that from 10% to 20% of all patients with chronic ulcer have one hæmorrhage or more.

The most suitable operations to contemplate are briefly:

1. Gastro-enterostomy.
2. Excision of the ulcer by cautery or knife *plus* gastro-enterostomy.
3. Sleeve resection or some other modification of the Billroth I operation.
4. Partial gastrectomy: (i) Billroth II operation, (ii) Moynihan's operation, (iii) Balfour's operation, (iv) Pólya's operation, (v) long loop gastro-jejunostomy with jejuno-jejunostomy.
5. Jejunostomy.

The value of these different procedures will be demonstrated by slides.

From the foregoing expression of evidence it is expected you will all be convinced that with surgical treatment of gastric ulcer there is one essential: wide excision, if possible.

It has been my practice over many years as a member of the honorary staff of this hospital (I reached my majority last September) to dispense with clamps. I have seen most stomach clamps tried, or have tried them myself, and not one has appealed to me as giving the essentials—uniform control of hæmorrhage without undue bruising of the bowel mucous membrane. The stomach mucosa is congested and invariably inflamed when ulceration is present, and in spite of what may be said to the contrary, a certain amount of bruising from their use is inevitable.

Stewart, of Leeds, showed me two *post mortem* specimens taken from cases in which gastric ulcers had been removed several months previously by eminent surgeons, and death had occurred later from intercurrent trouble with pronounced ulceration along the clamp line.

This is probably a more frequent sequel to gastro-enterostomy and gastrectomy when clamps are used than is appreciated—is in fact never looked for—and must be an important hidden factor in unsatisfactory results.

No resulting post-operative condition due to possible contamination from the escape of stomach contents or blood by not using clamps has been noted, and the method is confidently recommended as worthy of adoption. It is not so spectacular or so tidy to look upon, but the patients do well and, above all, one can be certain of absolute hæmostasis. The technique will be demonstrated fully by slides.

Perforations should be treated by simple suture, and if it is not possible to close them by suture they should be plugged by a piece of fat of the small or great omentum. In late perforations, when there is any food contamination, drainage is necessary in the kidney pouch and suprapubically, and all *débris* should be mopped up. With the experience of considerably more than one hundred cases, gastro-enterostomy has not been found necessary. All food should be withheld *per os* for the first few days, but fluid should be administered *per rectum* and intravenously in abundance.

Hæmorrhage.—Now a few words about hæmorrhage from ulcer, which is much discussed. Hæmorrhage from the stomach is a tragic spectacle for both physician and surgeon, and critical for the patient. For a fuller study of the subject you are referred to a masterly dissertation by Gordon Taylor.[23] His figures from 1919 to 1926 are: 32 patients operated upon, 6 died, a mortality rate of 18·75%. From 1926 to 1934 there were no operations; all the patients died. With regard to these latter figures, it is assumed that he saw only those patients who were *in extremis*.

Gordon-Taylor[34] again further states that if operation is contemplated there should be abundant facilities for repeated transfusions, and that operation should not be undertaken later than forty-eight hours after bleeding has ceased. I have seen many patients die from gastric hæmorrhage, and though I present to you my one and only patient operated upon, and, I think, saved by operation, it is my opinion that fewer people will die after medical than after surgical treatment. In any case operation should not be contemplated unless a recent X ray examination gives definite evidence of an accessible ulcer.

In this case of W.McK., aged thirty-four years, we had the definite history of ulcer, with X ray confirmation of ulcer of the lesser curve. It is now four years since operation, which was carried out under local anæsthesia. The ulcer was excised; transfusion was given immediately afterwards. The patient was almost unconscious when operated upon. Later, gastro-enterostomy was performed.

The patient thus furnishes an example of what can be done by wedge resection of ulcer with gastro-enterostomy. As you will see, he enjoys the best of health and takes any food.

Gastro-Jejunal Ulcer.—Balfour points out that the occurrence of gastro-jejunal ulcer after gastro-enterostomy for a gastric ulcer is rare, but I feel it is incumbent upon me to say a few words about it. Statistics combed throughout the Anglo-Saxon world estimate its incidence at about 2%. Moynihan's figures are under 2%, and they occur after gastro-enterostomy for duodenal ulcer, and I

present such a patient who, when first consulting me, still had his duodenal ulcer and extensive ulceration at the artificial stoma as well.

Radiography rarely displays a shadow of the ulcer, though it helps by disclosing delay in emptying through a narrow stoma and uneasy peristalsis in the upper part of the jejunum.

Symptoms are hunger pain relieved by food and alkalis one hour after food. Pain is generally to the left of the navel and may radiate to the groins. Tenderness and a tumour may sometimes be felt over the artificial stoma. Melæna and occult blood should arouse suspicion. As a rule wasting and anæmia are marked.

If a gastro-colic fistula has formed, there will be diarrhœa, foul eructations, fæcal vomiting and the passage of undigested food *per anum*. A barium enema will flow into the stomach.

If no serious complications are present, medical treatment should be given an extended trial, and, as a last resort, operation must be undertaken.

Radical operation is difficult and the mortality is high.

The various procedures adopted are: (i) excision of the ulcer, (ii) excision of the ulcer and abolition of the stoma, (iii) abolition of the stoma *plus* gastro-duodenostomy, (iv) partial gastrectomy, (v) closure of gastro-colic fistula by excising portion of the colon invaded and then attacking the ulcer.

Operations Depicted in the Illustrations.

Figures I to VIII demonstrate the steps of the operation of posterior gastro-enterostomy.

Figures IX to XII demonstrate those of resection for ulcer in the lower half of the stomach.

Figures XIII to XIX demonstrate those of partial gastrectomy.

The drawings of Mr. Farrell, carried out from actual work in the operating theatre, need but few explanatory notes.

For the guy sutures the finest of silk is used (when finally cut the small knotted portion left gives further security to the anastomotic line). For the outer peritoneal suture number 0000 lightly chromicized catgut is used.

For the second peritoneo-muscular and the innermost mucosal sutures number 0 plain catgut is used. On opening the stomach all fluid contents are drawn off by the sucker (likewise before the final closure any blood that may have trickled in during operation). The patient thus commences post-operative life with a clean stomach.

Conclusions.

The conclusions that we draw, then, are:

1. That gastric ulcer at its inception is a medical complaint.

2. That as gastric ulcers heal and tend to remain healed, and may never again cause symptoms, medical treatment should be given an extended trial.

3. That failure of medical treatment is to a great extent due to lack of cooperation on the part of the patient, and when it fails there should be no delay in recourse to surgery.

4. That expert medical treatment has a mortality rate approximating or perhaps higher than surgery in capable hands.

5. That there is a definite relationship between gastric ulcer and gastric cancer.

6. That with the medical treatment of hæmorrhage there is quite a high mortality rate.

7. That the surgery of hæmorrhaging ulcer is still in its infancy and with our present knowledge its routine treatment is safer in the hands of the physician.

8. That in hæmorrhage operation should never be contemplated unless there is abundance of blood for transfusion and definite X ray evidence of an accessible ulcer.

9. That wide excision is the most favourable operation, partial gastrectomy being advisable in the majority of cases.

10. Before operation careful preparation of the patient should be carried out, particular attention being directed to all sources of sepsis, such as from the appendix, teeth, tonsils, sinus *et cetera*.

11. After operation a diet should be given for at least three months, articles likely to over-stimulate the secretion of gastric juice being particularly avoided.

12. That in perforations some simple procedure should be carried out as expeditiously as possible, for the longer the delay after perforation, the higher the mortality rate.

References.

(1) J. B. Moynihan: "Some Problems in Gastric Ulcer", *The British Medical Journal*, December 8, 1928, page 1021.
(2) D. P. D. Wilkie: "Coincident Duodenal and Gastric Ulcer", *The British Medical Journal*, September 11, 1926, page 469.
(3) Arthur F. Hurst: "New Views on the Pathology, Diagnosis and Treatment of Gastric and Duodenal Ulcer", *The British Medical Journal*, April 24, 1920, page 559.
(4) W. Langdon Brown: "Predestination in Disease", *The British Medical Journal*, March 22, 1930, page 525.
(5) E. C. Rosenow: "Focal Infection and Elective Localization of Bacteria in Appendicitis, Ulcer of the Stomach, Cholecystitis and Pancreatitis", *Surgery, Gynecology and Obstetrics*, July, 1921, page 1.
(6) B. N. Berg: "Peptic Ulcers: Comparative Frequency after Deprivation of Bile and Pancreatic Juice", *Archives of Surgery*, June, 1934.
(7) C. H. Miller: Quoted by Langdon Brown, *loco citato*.
(8) Harvey Cushing: "Peptic Ulcers and the Interbrain", *Surgery, Gynecology and Obstetrics*, July, 1932, page 1.
(9) Howard Bullock: "Observations on Some Cases of Acute Ruptured Ulcers of the Stomach and Duodenum", THE MEDICAL JOURNAL OF AUSTRALIA, January 8, 1932, page 22.
(10) Ian Aird: "Perforation of Carcinoma of the Stomach into the General Peritoneal Cavity", *The British Journal of Surgery*, January, 1935, page 545.
(11) W. E. Gatewood, O. H. Gaebler, E. Muntwyler and V. C. Myers: "Alkalosis with Peptic Ulcer", *Archives of Internal Medicine*, July, 1928, page 79.
(12) Charles Bolton: In discussion on the prognosis of peptic ulcers, *Proceedings of the Royal Society of Medicine*, January, 1934, page 225.
(13) Josias J. Conybeare: *Proceedings of the Royal Society of Medicine*, January, 1934, page 228.
(14) D. C. Balfour: "The Case against Gastro-Enterostomy, etc.", *Collected Papers of the Mayo Clinic*, 1924, page 60.
(15) J. B. Moynihan: "Problems in Gastric Surgery", *The British Medical Journal*, December 8, 1928, page 1021.
(16) A. M. Cooke: In discussion of prognosis of peptic ulcers, *Proceedings of the Royal Society of Medicine*, January, 1934, page 238.
(17) Arhtur F. Hurst: Schorstein Lecture on Carcinoma of the Stomach, *The Lancet*, November 16, 1929, page 1023.
(18) A. J. Walton: "Carcinoma of the Stomach", *The British Medical Journal*, May 25, 1929, page 939.
(19) Victor Pauchet: *"Extrait des Neoplasms"*, Volume VI, 1927, Number 3.

(20) W. L. A. Willbrock: *Archives of Pathology*, Volume VIII, page 735.
(21) M. J. Stewart: "Gastric and Duodenal Ulcer", 1929, page 364.
(22) W. J. Merle Scott: "Relationship of Carcinoma and Callous Gastric Ulcer", *Surgery, Gynecology and Obstetrics*, February, 1928, page 199.
(23) G. Gordon-Taylor: Discussion on hæmatemesis, *Proceedings of the Royal Society of Medicine*, September, 1934, page 1524.
(24) Keith Ross: "The Treatment of Hæmorrhage from Peptic Ulcers", THE MEDICAL JOURNAL OF AUSTRALIA, February 8, 1930, page 168.
(25) Stewart: Quoted by Abrahams, *Proceedings of the Royal Society of Medicine*, September, 1934.
(26) John H. Watson: "Acute Perforating Duodenal and Gastric Ulcer", *The British Medical Journal*, August 2, 1930, page 169.
(27) J. B. Moynihan: Quoted by Watson, *loco citato*.
(28) H. Searby "The Treatment of Perforated Gastric and Duodenal Ulcers", THE MEDICAL JOURNAL OF AUSTRALIA, February 15, 1930, page 202.
(29) Donald Balfour: "Principles of Gastric Surgery", *Surgery, Gynecology and Obstetrics*, February, 1935, page 257.
(30) Norbert Ortner: "Abdominal Pain", 1922.
(31) Arthur F. Hurst: "Clinical Importance of Achlorhydria", *The British Medical Journal*, October 13, 1934, page 665.
(32) Arthur Connell: "Peptic Ulcer" (Editorial), *Surgery, Gynecology and Obstetrics*, March, 1933, page 710.
(33) L. R. Broster: "Gastric and Duodenal Ulcer", *The British Medical Journal*, November 3, 1928, page 786.
(34) G. Gordon-Taylor: "Bad Surgical Risks", *The British Medical Journal*, October 27, 1934, page 755.

Reports of Cases.

CHOLELITHIASIS IN A CHILD OF THREE YEARS AND EIGHT MONTHS.

By WOLFE S. BROWN, M.B., Ch.M. (Sydney),
Honorary Surgeon, Balmain and District Hospital.

HAVING reported a case of gall-stones in a child of nine years (THE MEDICAL JOURNAL OF AUSTRALIA, October 9, 1926) and finding that the case had aroused some interest, I now report a similar condition in a still younger subject.

W.McW. was a male premature child weighing 3·86 kilograms (eight and a half pounds), delivered by induction of premature labour on June 21, 1931. The child became robust and progressed without incident until February 27, 1935, when at 5 a.m. he screamed and complained of acute abdominal pain. He was brought to the surgery at 9 a.m. with all the signs of an "acute abdomen". He was carried in by his mother, being doubled up and unwilling to sit up straight. His temperature was 36° C. (97° F.) and the pulse rate 120 per minute. The abdomen was without movement, the muscles of the right side were on guard and acutely tender, the greatest tenderness being over the gall-bladder area. The symptoms suggested a diagnosis either of acute appendicitis or acute cholecystitis of obstructive type; the former was more probable on account of the child's age.

He was immediately admitted to the Balmain Hospital for operation. A small gridiron incision was made in the usual position in the right iliac fossa, by which a normal appendix was demonstrated and removed. Free clear fluid was present in the abdomen. The finger was now passed upwards and a tense gall-bladder, obviously the cause of the condition, was palpated. A right paramedian incision was now made. The tense gall-bladder was aspirated and turbid fluid without any trace of bile was withdrawn. A small stone was felt, impacted in the cystic duct, and after much manipulation was forced into the gall-bladder and removed. As the obstruction had been removed (bile was already showing in the gall-bladder), it seemed unwise to remove the gall-bladder from so young a subject; therefore a tube was stitched into the gall-bladder and the wound was closed. The tube drained bile freely, at first not so deeply coloured, but after a few days of the usual colour. The tube was removed on the tenth day and the wound healed rapidly.

On March 31, 1935, the child was up preparatory to being discharged from the hospital and apparently quite well; but on April 1, 1935, at 6 a.m., shivering and vomiting, with raised temperature ushered in erysipelas. This was unexpected. The site of the infection was on the abdomen just above the appendix incision and spread up the abdomen as far as the ribs and down the right leg half way to the knee. However, the erysipelas promptly subsided under the usual treatment, and the patient left the hospital quite well on April 15, 1935.

A SHARK ATTACK.

By FRANCIS C. CROSSLÉ,
Bulli, New South Wales.

AN unusual accident occurred to a young man on February 14, 1935. Whilst beginning to "shoot" a breaker at Austinmer on a pneumatic surf board, he felt a sharp pain in his right groin. At the time he was over a hundred yards from the shore and fortunately the board carried him in. He was conveyed to the surf shed and later to the Bulli District Hospital. There it was safe to investigate the nature of his injury and it was discovered that a long laceration extended downwards from below the mid-point of the groin for a distance of 20 centimetres (eight inches) to the inner aspect of the thigh. The long saphenous vein was severed, the pectineus, the *adductor longus* and the sartorius muscles were lacerated and the femoral artery was exposed. Two muscular branches were severed within 1·25 centimetres (a quarter of an inch) of the main artery. The wound was full of sand and it was found impossible to remove it completely. In addition there were two small wounds internal to the main wound and four small superficial wounds of the scrotum. On the posterior aspect of the thigh, at the level of the gluteal fold, there were eight small wounds extending horizontally through the skin and deep fascia. The longest was 7·5 centimetres (three inches) and the greatest distance apart was 5·0 centimetres (two inches).

It is remarkable that the wounds healed without any complication. Evidently sea-sand of an uncontaminated beach is comparatively unirritating. The patient's only complaint is impaired sensation over the inner side of the leg and foot.

Mr. David G. Stead has suggested that a white pointer shark probably caused the wounds.

ENTEROGENOUS CYST IN AN INFANT.

By KEITH ROSS,
Geelong.

ENTEROGENOUS or "mesenteric" cysts are rare. They have been discussed in several papers, notably that of Evans,[1] within recent years, but the notes of a new case may be of interest.

Case Report.

A.S., a male infant, six weeks old, was seen on September 6, 1934. The history was that nothing untoward had been noted till the child was circumcised at the age of three weeks. Since then he had gone down hill, with frequent attacks of colic and vomiting. The stools had remained normal. His weight had fallen from 3·15 kilograms (seven pounds) at birth to 2·82 kilograms (six pounds six ounces). An abdominal tumour had been noted at intervals within the last few days.

On examination a cystic tumour, 5·0 centimetres (two inches) in diameter, was felt in the right iliac fossa, and a second one of similar size below the left costal margin. Under anæsthesia the cystic tumours appeared to be joined by a solid middle part. Nothing else of significance was detected.

The cyst is sausage-shaped, measures 12·5 by 5·0 centimetres (five by two inches), and its wall has a resemblance to that of the intestine.

Acknowledgement.

I am indebted to Miss Wischusen, Melbourne Hospital, for the photographs.

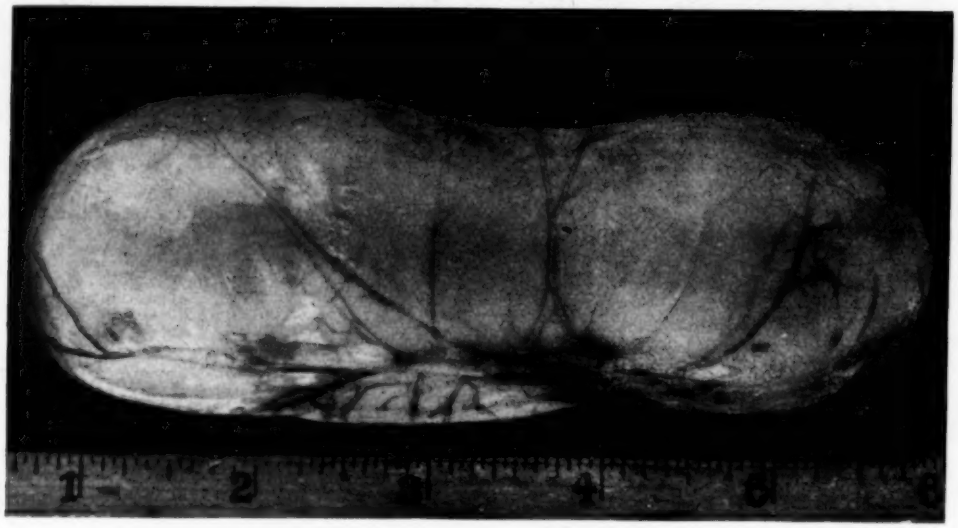


FIGURE I.
Enterogenous cyst.

Laparotomy disclosed a large, tense, thin-walled cyst springing from the mesentery in the region of the ileo-caecal junction and passing behind the ileum diagonally

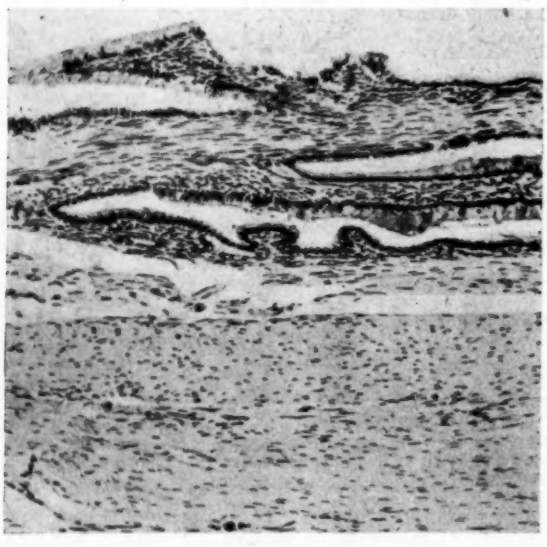


FIGURE II.
Cyst wall.

across the abdomen to the left hypochondrium. The cyst was removed and the child made a good recovery.[1]

[1] The child died suddenly four months later when apparently in perfect health. Early one morning it woke, either coughed or vomited up some blood, and died in a few moments.

Reference.

(1) Arthur Evans: "Developmental Enterogenous Cysts and Diverticula", *The British Journal of Surgery*, Volume XVII, July, 1929, page 34.

Reviews.

PREVENTIVE MEDICINE FOR THE PUBLIC.

MR. RITCHIE CALDER, a well known journalist, has written a volume on preventive medicine which should be stimulating to the more intelligent of the reading public.[1] He has put his finger upon the faults which still exist in the administration of public health ordinances; he notes, as we do here, the alarming spread of cancer, as revealed by statistics, the falling birth rate, and the doleful toll among child-bearing mothers, which seems to be as big a problem in Britain as in Australia. Mr. Calder was not only privileged to issue his book with the approval of Professor J. B. S. Haldane, but, whilst writing it, he sought the advice of such people as Sir Arthur Newsholme, the celebrated Chief Medical Officer to the Local Government Board, Dr. Edward and Dr. May Mellanby, Professor Langdon Brown, and many other significant figures in medicine. The result is a small book dealing intelligently with such problems as those of slum and factory life, diet, insanity, child welfare, and the possibilities of modern surgery, and in such a manner as to attract students of sociology and all interested in preventive medicine.

[1] "The Conquest of Suffering", by Ritchie Calder, with an introduction by Professor J. B. S. Haldane, F.R.S.; 1934. London: Methuen and Company, Limited. Crown 8vo., pp. 166. Price: 5s. net.

The Medical Journal of Australia

SATURDAY, JULY 13, 1935.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE MELBOURNE MEETING: AN APPEAL.

INCREDIBLE though it may seem, there are still some members of the Branches of the British Medical Association in Australia who have not awakened to the fact that the British Medical Association is holding its annual meeting in Melbourne in September. Even if they know that a gathering is to take place, some members speak of a "B.M.A. Congress" or even of a "Victorian Centenary Congress". That the significance of the meeting has not been grasped is causing some embarrassment to the Executive Committee and not a little concern as to the successful issue. Perhaps it may be useful to set out *seriatim* several points that need emphasis:

1. The meeting is not a congress; it is the annual meeting of the Association.

2. Every member of the Association is, in virtue of his membership, entitled to attend the meeting.

3. No membership fee is payable.

4. The meeting will commence on Monday, September 9, and last till Friday, September 13.

5. It is necessary for the Executive Committee to know the names of those who intend to be present.

In regard to the last of these points, it should be quite unnecessary to state that unless the Executive Committee knows how many visitors will be present it will be unable to make adequate arrangements for entertainment. The names of overseas visitors are known; members of the Australian Branches at least owe it to those who will act as hosts to give suitable notice of their intentions. We appeal to every member who intends to be present at the meeting to write without delay to Dr. J. P. Major, Local General Secretary, at 12, Collins Street, Melbourne. We also invite members who read these words to bring them to the notice of their medical friends who may not chance to see them.

The attention of readers is directed to certain alterations in and additions to the programme that appear elsewhere in this issue. As further items of information are available they will be published. The President-Elect, Sir James Barrett, and the members of the Executive Committee have the arrangements well in hand and are sparing no effort to make the meeting worthy of the occasion. Members of the Australian Branches will have the opportunity of meeting and of hearing discourses from distinguished visitors from the Old Country. Many of the leading visitors are set down to take part in the discussions of the several sections, but additional interest will centre round the clinical addresses that have been listed for the early mornings before the work of the sections starts. Each of the lecturers, Professor Edwin Bramwell, Professor E. W. Hey Groves, Mr. H. S. Souttar, Sir Ewen Maclean, Dr. Robert Hutchison, is known to Australian practitioners by writings and reputation. The opportunity of hearing them expound their views has but to be mentioned to be appreciated. Members are reminded that generous concessions are obtainable on the fares for rail and steamship travel; and that in each instance it will be necessary to produce a voucher signed by the Secretary of the Branch from which the member travels. Australian medical practitioners will regret neither the effort nor the sacrifice that they may make to take an active part in what will be a memorable event in the history of Australian medicine.

Current Comment.

PNEUMOCOCCAL PNEUMONIA.

PROBABLY no other acute disease has been more intensively studied by clinician, bacteriologist and experimentalist than has pneumonia. The last five years have seen an added impetus given to the study of this disease as the direct result of the introduction from America of Felton's concentrated serum. Statistical studies of the influence of this remedy on the mortality of the disease have been abundant, usually following the simple plan of administering the serum to every alternate patient after typing of the organism. Cecil's classical studies from the Bellevue Hospital, New York, showed a general mortality of 33·9% among 1,849 patients. English hospital figures vary from 19% to 27%, and it is generally agreed that the European death rate from pneumonia is distinctly lower than that of the United States of America. Sufficiently large numbers to be comparable to the above are not yet available for Australia. Dissection of Joule's Birmingham figures makes plain how mortality increases with age. Thus, among 542 patients whose ages range between ten and fifty years, the death rate was only 13%. Of 418 patients between ten and forty-nine years of age, only 8·6% failed to survive. Also, mortality figures are nowadays always correlated with type. Cecil, Baldwin and Larsen, also Blake, state that the type of pneumococcus responsible for pneumonia under the age of forty is Type I in from 40% to 80% of instances. Commenting on this finding, D. T. Davies, H. G. Hodgson and L. E. H. Whitby make a significant statement: "If we are to regard pneumococcal pneumonia as being the commonest form to attack those under forty years of age, then the period of life most favourable for spontaneous recovery corresponds to the incidence of the type most amenable to serum therapy." These authors believe that the specific type of pneumococcal infection can often be recognized clinically as well as bacteriologically.[1]

They have undertaken, with the support of the Royal College of Physicians of London, a detailed investigation of 184 patients treated at the Hammersmith Hospital, where, incidentally, the New British Post-Graduate School is situated. The Type I pneumococcus was responsible for 40% of the infections in their series, and the majority of their affected individuals were under the age of forty. The illness of 19 of these 55 patients was deemed serious enough for them to have serum treatment.

Analysing available figures, the authors conclude that the English mortality from Type I pneumonia cannot exceed 10% to 15%. Clinically the course of Type I pneumonia corresponds to the classical text book description. The average age of their group was thirty. Termination was usual by early crisis after a clear-cut onset, followed by high sustained fever: in short, the pneumonia of the "healthy" young adult. Leucocytosis was abundant, with a rapid fall at the crisis. Agglutinins were detectable, not just before or at the crisis, but two or three days afterwards, and disappeared in a fortnight, unless a complication was brewing. Davies and his associates deduce that the presence of agglutinins is directly associated with the process of resolution, and that they are not an index of immunity. Sixteen patients of the Type I series were treated with serum; five on the second day, five on the third, and six on the fourth; and all because the symptoms were severe or the "risk" was poor. Two infections ended fatally, one from meningitis, the other from bilateral empyema. In both the blood was sterile, while four patients showing a positive blood picture recovered. The symptomatic effect of the serum was undoubted. Though this was more evident when the serum was given in the first three days, some effect was obtained when it was given later. The investigators consider that serum should not be withheld from patients desperately ill in the sixth or seventh day. If life could be thus prolonged for a further day, it might be possible for a crisis to be established. All patients showed an immediate reaction to the serum, characterized by sweating, tachycardia and increase of general distress. Serum sickness appeared in six cases between the ninth and sixteenth days.

Concerning Type II pneumonia, usually associated with a higher mortality than Type I, these authors found the English death rate to be lower than the American. Bacteriæmia is far more common, toxæmia is greater, termination by crisis is unusual, and there are fewer complications. No agglutinins can be detected until resolution occurs. None of their patients was admitted to hospital sufficiently early for serum treatment. With or without serum Type II still remains a serious disease. Type III has always been regarded as the most serious of all. In Cecil's series the incidence of this type was 11·1%; in this series 15%. Doubt has, however, been thrown on the virulence of the organism; the high mortality is due more probably to the fact that the elderly are more frequently attacked.

The incidence of Type IV pneumonia is generally placed at 30%. Recent typing, in Sydney at least, shows that the majority of pneumonias are of Type IV group. There is no general agreement about the mortality rate. Twelve patients in this series are placed under the heading "Anomalous and Mixed Infections" and illustrate the fallibility of sputum examination alone. Apparently there are three sources of error: (*a*) Pneumococcal pneumonia may have streptococcal complications. (*b*) Patients may be diagnosed as suffering from a type of infection which later proves to be of a different type. (*c*) In some patients the sputum contains more than one type of coccus. Study of the precipitins, agglutinins and protective substances should help to decide some of these problems. The study of agglutinins is most disappointing. Their presence is not remarked until resolution sets in; they are no indication that a patient will be free from relapse or complication; and are certainly not an indirect indication of type, as suggested by Sabin.

[1] *The Lancet*, April 6 and 13, 1935.

The examination of a case of pneumonia is incomplete nowadays without the typing of the infected pneumococcus. Cecil, who has had an enormous experience of pneumonia and serum therapy, remarks, with significance, that it is more important to know the type of pneumococcus in a case of pneumonia than it is to know the type of organism responsible for an enteric fever. Experience and good laboratory equipment are essentials. It is unfortunate that the procedure is so much more difficult in the first two or three days than later, because of the scanty sputum and impure culture. Nevertheless one important lesson to be learned from this work is that, within limitations, the organism responsible can usually be identified, as shown by the results of specific therapy, and also that some benefit can be expected from serum given as late as the fifth or sixth day to the severely ill sufferer.

To treat all patients suffering from lobar pneumonia with Felton's serum is unnecessary and costly. By no means all Type I infections need to be treated by serum, for the mortality of the untreated patient is low. Serum should be reserved for patients over the age of forty, with long-standing chest disease, arterial hypertension and a leucocytosis below 15,000 per cubic millimetre. Repeated leucocyte counts form one of the best methods of following the progress of the disease. It is refreshing to learn that the patients in the London series who had a positive blood culture came through as well as those without. A combined research in a number of hospitals on this problem is urgently required in Australian capital cities in order that we may share to the full in the undoubted benefits of Felton's serum. Such an inquiry could well follow the plan adopted by Davies and his colleagues.

THE RADIOLOGY OF PNEUMONIA.

THE suddenness of onset, unsuitability of standard apparatus and the gravity of an acute pneumonia have had the result that this disease has only rarely been adequately followed by radiographic visualization. By using a powerful machine requiring a very short exposure and a tube-stand six and a half feet above the patient's chest, Daniel Davies, Graham Hodgson and L. Whitby have succeeded in making a remarkably good series of 1,500 radiograms in 119 cases of pneumococcal pneumonia.[1] The patients were simultaneously the subject of careful clinical, serological and bacteriological examination, so that correlation has been possible between all these aspects of the illness. Various well established clinical facts and hypotheses have thereby received confirmation, while some entirely new facts have been elicited. To interpret these pictures aright, one requires a clear knowledge of the position of the interlobar fissures and a realization that, in considering the various densities of the shadow, one must remember the wedge-like shape of the lobe. Lateral skiagrams give great assistance in this regard. Radiographic consolidation takes place very rapidly in pneumonia, the characteristic appearance being usually well marked throughout the entire lobe within twenty-four hours of onset. In most the first change is an increase in the density and extent of the hilar shadow on the affected side. Coincidently three other changes, namely, an elevation of the diaphragm on the affected side, a narrowing of the overlying intercostal spaces and occasionally some deflection of the mediastinum towards the pneumonic area. These appearances would seem to give some support to the theory of lobar collapse, although these authors maintain a neutral attitude towards this question. The opacity reaches its maximum density by the fourth or fifth day and remains so until resolution begins, the first sign of which is enlargement of the hilar glands. The "central" (that is, originating at the hilum) mode of spread is far commoner in adults than the "peripheral". The type of pneumonia present can frequently be predicted from its radiological appearance. In Type I these are distinct and uniform. Two such patients, examined by X rays within thirty hours of onset showed consolidation on both sides of the chest, though clinical signs had suggested unilateral involvement, and in general radiography showed more extensive involvement than was indicated by clinical signs, especially in upper lobar affections. The time required for resolution was very variable, considerable delay occurring where calcified hilar glands told of past chest disease. Dense consolidation was associated with severe symptoms. Empyema appeared only when the shadow was very opaque; here physical features are a more reliable guide. Only one patient in the Type I group showed any extension of consolidation after a crisis had occurred. No material hastening of resolution was observed in the serum-treated patients; indeed, some showed progressive consolidation even after clinical relief had been afforded by Felton's serum.

In Type II pneumonia the above findings were also noted, but there was a tendency for a slight but permanent increase in the pulmonary striæ to be seen afterwards. Type III pneumonia was not so homogeneous, the level of the diaphragm was unaltered, and more disparity was found between clinical and X ray signs in this group than in any other. In Group IV the opacity was only moderately dense and peripheral. In some of these patients resolution was extremely rapid. Observations were also made on the heart shadow, and the authors came to the conclusion that there was usually no enlargement to be detected. In a few patients showing a relative bradycardia and hypotension, however, an increase up to three centimetres was noticeable in the transverse cardiac dulness.

It is obvious that much assistance could occasionally be given the hospital clinician if X ray apparatus which would not disturb the patient could be made available, so that the exact extent of the disease or even its presence could be determined. There is little doubt, however, that the physical features and guides are usually sufficient to a skilled physician.

[1] *The Lancet*, April 20, 1935.

Abstracts from Current Medical Literature.

RADIOLOGY.

Bronchial Carcinoma.

PEDRO L. FARINAS (*American Journal of Roentgenology*, December, 1934) states that bronchography under Röntgenoscopic control permits a diagnosis of bronchial carcinoma in its early stages and should be used whenever a bronchogenic carcinoma is suspected on clinical grounds. A skiagram made after the injection of iodized oil has little value. In cases in which there are neoplastic changes which do not completely obstruct the bronchus, it is necessary to take a series of skiagrams at the best angle possible, so that any small alteration of the aspect and contour of the bronchus may be studied with certainty. The bronchographic aspect of bronchial carcinoma varies with the type of the tumour. In the polypoid form of tumours, showing a marked prominence in the lumen of the bronchi, the bronchographic shadow is a filling defect. When seen *en face* it appears as a negative shadow, round or irregular, due to the displacement of the oil by the tumour. When the defect is seen in profile, the shadow appears as an irregular notch in one border of the bronchus. This type of tumour is found mostly in the main bronchi and can be easily demonstrated; but when it is in the small bronchi its demonstration is very difficult, especially when other small filled bronchi are superimposed. In the infiltrating type the bronchographic shadow is that of an incomplete concentric stenosis with irregular borders, generally occupying the bronchi to a large extent. This type of tumour is usually seen in the main bronchi and extends along its branches. When it is found in small bronchi it is necessary to continue the injection in order to show the stenosis to its full extent; also the prestenotic dilatation, because the oil enters through the stenosis very slowly. When the process is more advanced the stenosis is complete and we can see the arrest of the iodized oil at a point. In some cases of bronchial cancer the walls of the bronchi become ulcerated and, instead of giving rise to an obstruction, the necrosis forms cavities of various size, but always irregular. Their bronchographic aspect is that of a cavity of irregular form that can be distinguished from annular bronchiectasis on account of its irregular contour. This type is usually seen at the hilus.

Congenital Cyst of the Lung.

JAMES L. DUBROW (*Radiology*, April, 1935) states that various theories have been advanced as to the ætiology of congenital cysts: (i) bronchial maldevelopment—a defective embryologic development of the bronchi resulting in complete or incomplete stenosis of the main bronchus or bronchi, usually of the upper lobe, with distal dilatation of the end branches; (ii) alveolar agenesis—defective embryologic development of the alveolar tissue, which leaves the bronchi unsupported and consequently causes them to become unduly dilated; (iii) defective embryologic development (*Anlage*) of the lymph vessel systems of the corresponding lung. The cysts may be filled with fluid if the deformed bronchus is completely occluded; if incompletely stenosed, they will contain air. Clinically the following conditions, amongst others, may be simulated by these cysts: (i) Bronchiectasis. The congenitally dilated bronchi and bronchioles may retain some mucosal secretions, and if the latter become secondarily infected it may be impossible to differentiate the congenital sacculations from the acquired form. (ii) Pulmonary atelectasis. The alveolar agenesis affecting a whole lung leads to its arrested development with consequent narrowing of the intercostal spaces, elevation of the diaphragm, and displacement of the heart and mediastinum to the affected side, which on the Röntgenogram appears as a unilateral opacity. Lipiodol injection will demonstrate extensive sacculations in contradistinction to acquired massive pulmonary atelectasis in cases in which the main bronchus is completely obstructed. (iii) Spontaneous pneumothorax: (*a*) localized, silent, in cases in which the cystic changes involve a whole lobe or the greater portion of the lobe and the congenital stenosis of the bronchus is such that ingress and egress of air are free, the cyst will contain air, and on the Röntgenogram it will resemble a localized pneumothorax (or perhaps cavitation); (*b*) valvular, in cases in which the congenital stenosis of the bronchus is such that egress of air is interfered with while ingress is free. The mechanism is that of obstructive emphysema, owing to a check-valve obstruction wherein with each respiration more air becomes incarcerated in the subjacent lung, which dilates and presses upon the mediastinum and the heart, displacing them to the unaffected side; the contralateral lung is thus encroached upon.

SAMUEL SCHENCK AND JACOB STEIN (*Radiology*, April, 1935) state that although the ætiology of congenital lung cysts is still obscure, the opinion prevails that we are dealing with a congenital malformation or developmental error, which results in a dilatation of the terminal bronchioles filled with a tenacious glairy fluid. If the cyst communicates with an adjacent bronchiole, this fluid is replaced by air and may remain stationary in size, provided the communication is patent during both phases of respiration. If the air is allowed to enter the cystic cavity and the communicating channel collapses or is closed off during the expiratory phase, the cyst enlarges or balloons, thereby producing severe attacks of dyspnœa and cyanosis, which often terminate in death. A similar mechanism obtains in the case of multiple or honeycombed cysts. The diagnosis is made chiefly from a complete radiological survey, and the condition must be differentiated from general or localized pneumothorax, lung abscess, encapsulated fluid, pneumonia, solid tumours and acquired cystic disease, such as bronchiectasis, echinococcic cyst and hydatid cyst. The prognosis is very grave, especially in infants, and only four patients are recorded to have made a complete spontaneous recovery.

Gonorrhœal Arthritis.

PETER J. KAPO (*American Journal of Roentgenology*, March, 1935) states that the character of gonococcal arthritis was first considered to be essentially monoarthritic; later it was admitted to have a polyarthritic onset with ultimate localization in one large joint; and now the gonococcus is alleged to be a frequent invader of the temporo-mandibular and sterno-clavicular joints, which latter joints are probably never involved in rheumatic fever. The protean manifestations of gonococcal arthritis necessarily simulate a wide variety of joint affections. Rheumatic fever is generally more amenable to salicylate therapy, occurs earlier in life and leaves no residual joint damage. Tuberculosis has a more insidious onset, does not flit from joint to joint, and usually produces decalcification without distortion of the bone structure. Spotty ground-glass atrophy of the bones neighbouring an arthritis is suggestive of a gonococcal origin, especially in the absence of any evidence of a serious streptococcal or pneumococcal infection. Bone erosions are frequently found in acute gonorrhœal arthritis. Sometimes a punched-out appearance may be simulated or the cartilage alone may be affected. Osteolysis and chondrolysis are typical expressions of the pathological changes found in an acute infectious arthritis, whether of pyogenic or gonococcal origin. The apparent overgrowth of bone at the edges of such joints may simply be a flaring effect due to pressure on the softened osseous structure. Ankylosis may be expected whenever there has been extensive destruction of the articular cartilage. Widening of the joint spaces may be found in an early intra-articular gonorrhœal effusion.

The "Phrygian Cap" in Cholecystography.

EDWARD A. BOYDEN (*American Journal of Roentgenology*, May, 1935) states that the apparent kinking or biloculation of the fundus of many gall-bladders is due to a congenital anomaly which results in a folding-over of the fundus into a shape resembling a "Phrygian cap". There are two primary types of folded fundus: (i) the concealed or retroserosal type, which is due to an early folding of the epithelial *Anlage* of the

gall-bladder behind the peritoneal investment of the embryonic *fossa vesicæ felleæ*, and (ii) the serosal type, which is due to aberrant folding of the fossa itself—a type in which the peritoneum follows the bend in the gall-bladder as the *pia mater* follows the convolutions of the brain. While in the second type the folded fundus is somewhat movable, nevertheless the bend is fixed by fœtal ligaments, by rudimentary septa or by congenital constrictions of the lumen.

PHYSICAL THERAPY.

The Röntgen Treatment of Pituitary Tumours.

GEORGE E. PFAHLER AND EDGAR SPACKMAN (*The American Journal of Roentgenology and Radium Therapy*, February, 1935) give the case notes of eighteen patients suffering from pituitary tumours, with all the special investigations and end results. They recommend Röntgen therapy as routine treatment after close consultation with surgeon and neurologist. They state that they have not seen any harmful effects. The dosage given is 1,600 *r* through three ports of entry. If this is not efficacious, surgery is indicated. Treatment takes up to two months and in no way interferes with surgical procedure. Fields of vision are examined monthly during treatment and the reaction period, and then at three-monthly intervals. Post-operative Röntgen therapy is strongly recommended in every case in which there is doubt of complete removal of the tumour. Best results have been obtained with the solid pituitary adenomata; cystic adenoma and other types of tumour responded less favourably. It is surprising to find the disease often so far advanced when the patient seeks medical advice.

Radiathermy in Medicine.

DISRAELI KOBAK (*Archives of Physical Therapy, X-Ray, Radium*, January, 1935) discusses radiathermy in medicine. Radiathermy represents an important advance in high frequency therapy because it favourably influences inflammatory and purulent affections which cannot be treated with diathermy and for which diathermy treatment is even dangerous. Radiathermy operates according to Joule's and Ohm's laws and influences biological structures by capacity effects instead of by conductivity, as is the case with diathermy. Greater capacity insures greater concentration of energy in deep structures. Radiathermy utilizes two regions of the Hertzian spectrum (30 to 12 and 12 to 3 metres), provokes distinct physiological changes, depending on the wave-length, and creates thermal, selective and specific reactions. While evidence points towards specificity, the striking therapeutic effects have been associated with heating phenomena, thus placing conclusions of specificity in the realm of theoretic speculation. The technique of radiathermy is essentially similar to that of diathermy, except that use is made of insulated special electrodes, which are held at varying distances from the skin of the parts to be treated, the clothing of the patient often remaining undisturbed during applications. Control of the current is not determined by an ammeter, as in diathermy, but presents no technical difficulties. The principal indications for radiathermy are virtually all affections for which diathermy has proved effective. In addition, radiathermy is capable of producing hyperæmia of osseous structures. It has much deeper heat effects than diathermy and is therefore the method of choice in deep-seated inflammatory processes. Its striking and revolutionary indication is its favourable influence on suppurative processes for which diathermy is distinctly contra-indicated. The large range of affections for which radiathermy has already proven pronouncedly alleviating and even curative promises it an important status in therapeutics.

Protracted External Irradiation in the Treatment of Neoplasms of the Mouth and Throat.

MILTON FRIEDMAN AND RIEVA ROSH (*Radiology*, January, 1935) present a two-year clinical study of the effect of protracted external irradiation on neoplasms of the upper respiratory tract. They compare γ and high voltage X rays, ascertain the optimum number of days required for the dose, the optimum number of hours per day, and they try to define the indications for interstitial irradiation. Techniques are given, three different types being used. The authors found but a slight difference between the primary effect of X rays on the tumour and the effect of γ rays. The erythema from a single erythema dose of radium rays appears at a much later date and is more prolonged than from X rays. Gamma rays produced a more profound effect upon the normal tissues around the tumour and rendered marked intolerance to subsequent interstitial radiation. The physical factors are that the maximum dosage over the same period causing the same intensity of biological effect of epidermitis and epithelitis is produced with 3·5 skin erythema doses with radium and 4·5 skin erythema doses with X rays. The duration of the period of administration of the treatment is probably the most important single factor found in protracted external irradiation. Each histological type of neoplasm has its own rhythm of response and must be paralleled by a suitable time duration for the treatments. Furthermore, each type of ray quality has a characteristic period of greatest effectiveness during which the destructive phase is in the ascendancy and the healing phase has not yet commenced. The proper delicate adjustment of these two factors is essential to efficient protracted irradiation. The authors found that the double small 100-milligramme pack, producing continuous irradiation for twenty-four hours a day, is an efficient therapeutic medium which closely rivals the large five-gramme pack. It appears to produce the same effect upon the skin and mucosa with only four-fifths of the dose given with the larger pack.

Malignant Disease of Larynx and Pharynx.

A. ZUPPINGER AND R. STEWART-HARRISON (*Journal of Laryngology and Otology*, November, 1934) publish a second communication, two years after the first, on the subject of malignant disease of the larynx and pharynx. The authors are convinced that they have obtained an improvement in subsequent cases and that they shall continue to attain improvement by attention to the following points: general condition of the patient, suppression of distant metastases, advance in the treatment of recurrences, more particularly of residual tumours, and, above all, in the treatment of glandular metastases. The tube voltage has been increased, as also has the filter used. The total dose now reaches 700 to 800 *r* (international). Two treatments are given daily for five days a week. One hundred and fifty-nine patients are considered. Of these, 120 completed treatment. At the present moment 27 are living free from all disease two and a half to five years after treatment. Nine died of intercurrent disease, free from symptoms locally, during the first half year after treatment. Fourteen died of intercurrent disease more than one year after treatment. In 25 cases distant metastases developed by freedom from local and regional disease. Fifteen recurrences were seen, only one of which occurred after the first year. After an observation period of two and a quarter years the local success is 52%, the relative success 26%, and the absolute success 17%. The authors accept protracted fractional treatment as the treatment of choice in all cases of malignant disease of the pharynx and in most cases, if not all, of carcinoma of the larynx.

Malignant Tumours of the Kidney in Children.

ERNEST A. POHLE AND GORDON RITCHIE (*Radiology*, February, 1935) report six kidney tumours occurring in children under the age of six years. Pathology is discussed, especially the loose application of the term "Wilm's tumour". Tumours of the kidney in children do not produce symptoms till they have reached considerable size. In contrast to hypernephroma, these highly malignant tumours seldom cause hæmaturia early in their course. Prognosis is extremely poor, mortality is certainly above 90%. The treatment advised is pre-operative irradiation followed by surgical removal of the residual mass and then post-operative irradiation. Case notes of the six cases are given and a discussion follows.

British Medical Association News.

ANNUAL MEETING.

THE ANNUAL MEETING OF THE WESTERN AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Hospital for the Insane, Claremont, on March 24, 1935, DR. R. H. CRISP, the President, in the chair.

Financial Statements.

Owing to the absence of the Honorary Treasurer, Dr. Donald Smith, the balance sheet and financial report for the twelve months were not presented. Consideration of these was deferred until the next general meeting of the Branch.

Dr. T. C. Boyd and Dr. L. A. Hayward were elected Honorary Auditors.

Office-Bearers.

The President declared the following office-bearers elected for the ensuing twelve months:

President: Dr. F. L. Gill.
President-Elect: Dr. A. Syme Johnson.
Past President: Dr. R. H. Crisp.
Honorary Treasurer: Dr. Donald Smith.
Honorary Secretary: Dr. L. E. Le Souef.
Members of Council: Dr. F. W. Carter, Dr. J. J. Holland, Dr. M. K. Moss.

Annual Report.

The President read the annual report, as follows:

Membership.

Membership of the Branch has increased from 258 to 260.

Deaths.

I regret to report that during the year the following practitioners have died: Dr. R. S. Andrews (Greenbushes), Dr. E. Black (Perth), Dr. W. T. Hodge (Derby), Dr. Simon Joel (late Bunbury), Dr. L. J. Robertson (Subiaco).

Meetings.

There were nine general meetings during the year, with an average attendance of 36.

The annual meeting was again held, by the kind invitation of Dr. James Bentley, at the Hospital for Insane, Claremont.

Clinical meetings were held at the Children's and the Fremantle Hospitals. Both were kindly arranged by the superintendents of these hospitals.

Interesting papers were read by Dr. R. G. Williams, Dr. L. E. Le Souef, Dr. H. G. D. Breidahl and Dr. Cook.

A special meeting was held jointly with the legal profession on the subject of sterilization, there being present twenty-six legal practitioners and 42 medical practitioners.

A successful post-graduate meeting was held during September, when Dr. H. C. Trumble and Dr. R. P. McMeekin, from Melbourne, both attended and lectured during the week.

The annual dinner was held at the Imperial Hotel, when fifty-one members were present. The dinner proved a thorough success.

Council Meetings.

The Council met twelve times, members attending as follows:

	Meetings.		Meetings.
Dr. Atkinson	8	Dr. Holland	9
Dr. Crisp	11	Dr. Le Souef	11
Dr. F. Gill	10	Dr. M. K. Moss	12
Dr. H. B. Gill	7	Dr. D. Smith	12
Dr. H. J. Gray	12		
Federal representatives:			
Dr. Paton	10	Dr. McWhae (for Dr. Hadley)	7
Dr. Hadley	0		

Federal Council.

Dr. Hadley, one of our representatives to the Federal Council, resigned, and for 1935 Dr. Paton and Dr. McWhae have been appointed representatives. I desire, on behalf of the Council, to express our appreciation of Dr. Hadley's work.

London Representative.

Sir Thomas Dunhill has been elected by this Branch for the years 1935-1938.

Maternal Mortality.

This matter is being taken up by the Federal authorities with a view to obtaining Australia-wide information and recommendations. State and Federal conferences are being held. Our delegates appointed by the Council for this work are Dr. D. P. Clement and Dr. G. A. Thompson.

Library.

During the year the library has made great progress in the hands of Dr. Cyril Bryan, the Honorary Librarian. A typist has been supplied and the telephone has been installed. Excellent work has been done in cross-indexing the journals supplied to the library. It only remains now for members of the Association to use the library for it to be a success.

Mining Agreements.

Satisfactory agreements for medical services at Cue, Mount Magnet, Laverton, Payne's Find have been carried out.

Scheduled Fees for Police and Aborigines.

Separate schedules have been agreed to for medical fees for service on police officers and also on aborigines.

North-West Medical Service.

Under the guidance of a subcommittee consisting of representatives from the Australian Inland Mission, the Pastoralists' Association, the Medical Department, and representatives from this Association, the scheme of the North-West Medical Service has been duly installed. The Council has agreed to medical appointments north of Carnarvon being full-time appointments at a salary of £1,000 *per annum*, all the charges and fees for medical service being collected by the Medical Department. Our representatives, Dr. Holland and Dr. Paton, have done excellent work in this matter.

Workers' Compensation Act.

The work of our Special Workers' Compensation Act Subcommittee has been continued in the able hands of Dr. M. K. Moss and Dr. F. L. Gill, who have been available in all cases of disputed accounts and other matters arising out of the Act. They have done excellent work for the State and other individual insurance companies applying for an opinion.

This subcommittee was originally formed on the 6th April, 1927, to function, with a committee of the Underwriters' Association, as a joint committee to handle disputes and complaints arising out of the *Workers' Compensation Act*.

This joint subcommittee was disbanded at the instance of the Underwriters' Association at the end of 1931. Our committee, however, continued to function. The underwriters have again approached the Council to reform the joint committee. Conferences have been held and friendly relations have been reestablished between the two bodies, and we hope the joint committee will be able to lubricate the joints in the Act to the mutual benefit of all concerned.

A pamphlet has been issued by the Council on the whole subject of workers' compensation insurance, and it is hoped that all members will carefully peruse this document and loyally cooperate to make such a generous act the success it deserves to be.

Treatment of Sustenance Workers, Agricultural Bank Clients and Indigents.

The Council went to a lot of trouble to try to get some relief for medical men for their treatment of these classes of patient. The difficulty, however, is that the Government does not admit liability to supply medical treatment for the indigent. A scheme of contract practice for these patients was discussed. That part of the profession vitally interested was circularized on the matter more than once. Out of eighty inquiries of country doctors we only got fourteen replies. It proved impracticable for us to get the full statistical information needed or to reach any unanimity in the proposed scheme, so we were forced to temporarily abandon the proposed approach to the Government on this matter. We did ascertain, however, from the Medical Department that in the case of indigents who are treated in an honorary capacity, out-of-pocket expenses would be considered in any case put up to the Department.

Broadcasting.

The Council was approached by the Broadcasting Commission during the year with a view to the arrangement of a series of broadcast talks of "popular" nature on medical or pseudo-medical topics by members of the Branch. These talks are official in that they are previously censored by the Council, but they are given anonymously. Some of the subjects already used are as follows: "Medicine and the Allied Sciences", "A Medical School", "Community Hospitals", "Diphtheria Immunization", "St. John's Ambulance Service". Further titles will be: "Lodge Practice", "North-West Medical Service", "Hospital Policy". This is an entirely new departure as far as we are concerned in publicity. It gives the medical profession an opportunity to explain its attitude to various matters of general medical and medico-political nature, and provides us with an excellent medium for moulding popular opinion.

Information to Members.

The policy has been followed of informing members of matters of interest by putting paragraphs on the back of the monthly agenda papers. Many such matters about which members are already informed are not included in this report.

I would like to thank the members of the Association for the support they have given to me as President. I especially desire to thank members of Council for their loyal support during the year.

President's Address.

Dr. R. H. Crisp read his President's Address (see page 33).

Induction of President.

Dr. Crisp then introduced the new President, Dr. F. L. Gill, and vacated the chair in his favour.

Library Report.

Dr. Cyril Bryan, the Honorary Librarian, presented the library report as follows:

I beg to submit the report of the British Medical Association Library for the past year. Dr. Hislop and Dr. McKellar Hall having resigned as Joint Librarians at the last annual meeting, I was later appointed Librarian, with Dr. A. T. White as Assistant Librarian. At the same time a paid full-time library assistant was appointed.

The books constituting the library are without exception out of date, half of them being dated in the last century, and only a handful bearing a post-war date. Following, therefore, in the footsteps of my predecessors, we have concentrated on the medical journals, and a comprehensive index is made of all the contents of such medical journals as we can get hold of within a few hours of their receipt.

In a scheme propounded by Dr. Hislop and Dr. McKellar Hall a large number of medical and surgical journals were promised by our various colleagues, and on taking charge I wrote or waited on all these gentlemen, asking if they were still prepared to donate such journals as stood against their name. Some, however, had discontinued subscriptions to those journals for one reason or another, but the majority have handed their copies to us a week or so after they have received them. Of these, the following are received regularly and promptly indexed: *The Lancet* (Dr. J. Vere Arkle), *The Practitioner* (Dr. A. T. White), *Proceedings of the Royal Society of Medicine* (Dr. J. Vere Arkle), *The British Journal of Surgery* (Mr. J. P. Ainslie), *Journal of Otology, Rhinology and Laryngology* (Dr. A. Farmer), *The Australian and New Zealand Journal of Surgery* (Dr. M. K. Moss), *The General Practitioner* (Dr. J. Gordon Hislop), *South African Medical Journal* (Dr. J. O'Donnell), *Anæsthesia and Analgesia* (Dr. M. K. Moss), *The Journal of the American Medical Association* (Dr. Bruce Hunt), *Medical Clinics of North America* (Dr. G. W. Barker), *Surgical Clinics of North America* (Dr. G. W. Barker). These I have stated are regularly received. There are various other journals which do not come in regularly, but will do so soon.

I have to report that a number of the bound volumes of *The British Medical Journal* and *The Lancet* are missing. I hope that members will help me to trace them. I have done all I could so far.

The missing *British Medical Journals* are: Volume I, 1892–Volume I, 1897; Volume II, 1903; Volume II, 1905; Volume II, 1912; Volume II, 1916–Volume II, 1925.

The Lancets begin in October, 1847, and the missing volumes are: Volume I, 1850 to Volume II, 1871; Volume II, 1874, to Volume I, 1875; Volume I, 1877, to Volume II, 1877; Volume I, 1878; Volume II, 1893, to Volume II, 1895; Volume II, 1896; Volume I, 1916, to Volume II, 1916; Volume II, 1917, to Volume II, 1933.

From the beginning of the year all references to medical matters appearing in the two Perth daily papers are collected by the library assistant and filed for reference.

Up till December 31, 1934, there were exactly one hundred visits to the library. These do not represent visits from one hundred different members I hasten to add. From the beginning of the year to yesterday there were forty-five visits. I should add that it is almost uncanny how many of these visitors come to consult a volume of *The British Medical Journal* or *The Lancet* which happens to be missing. I would think it a joke if it did not happen so often, and it causes me to make every effort to have these bound volumes intact.

In concluding this report I wish to ask the cooperation of all members of the British Medical Association in securing photographs, instruments, documents, in fact anything that in any way touches on the history of medicine in Western Australia, or in Australia and the world generally. A library is of all places the proper repository of such relics, and I hope members will help me to secure such as are now available. Beginning with Flinders, who was the son of a doctor and who ran away to sea, in time giving us the very name our country bears, I have begun the collection of photographs of the old doctors in our early history, of the presidents of the British Medical Association since its formation here, of places and things which touch on our profession in any way. Dr. Lucraft has promised to photograph for me the first medical certificate issued in the colony; the first medical certificate of admission to the Colonial Hospital in 1830; the first application to practise here, that of Dr. Milligan; the first venereal disease certificate; all of which have been unearthed in the colony's archives. Dr. Kenny has promised me a water colour of the first hospital erected at Garden Island on the arrival of the *Parmelia* in 1829. I am also most anxious that this Branch of the British Medical Association will produce a copy of the wonderfully interesting diary of Dr. Viveash, a pioneer of the thirties; and also a copy of the letters written by Dr. Collie, the second colonial surgeon, in 1832-1835. It is only fitting that the library of the British Medical Association (Western Australian Branch) should include these historical documents which tell us so much of our early history and of the history of the profession in this colony and State.

Our thanks are due to Dr. T. C. Boyd for his gift to the library of five bound volumes of *The Proceedings of the Royal Society of Medicine*, and to Dr. A. Webster for a copy of his book, "Cardiac Arrhythmia and the Neo-cardiology"; also to the Victorian Branch of the British Medical Association for several urological monographs.

The report was received and adopted.

Discussion concerning the site of the library took place and the meeting asked the Council to reconsider the question of its site.

The Medical Benevolent Association of Western Australia.

The annual report of the Medical Benevolent Association of Western Australia for the year ended December 31, 1934, was presented by Dr. J. R. G. Maitland. The report is as follows:

For the year 1934 the list of members of the Medical Benevolent Association numbers 108, of whom 8 are life members and 66 financial members.

At the last annual general meeting a motion was carried that the subscriptions to the Medical Benevolent Association, instead of being paid direct to the Treasurer, should be paid to the Treasurer of the British Medical Association together with the usual annual subscription to that Association. It was thought that in this way more subscriptions would be collected and more members enrolled. It is too early in the year yet to decide if this alteration has had the desired results.

During the year benevolent contributions were made to the extent of £26.

The bank balance on December 31, 1934 (as per balance sheet) was £332 19s. 3d., or £22 in excess of last year.

The report was received and adopted.

Dr. Maitland appealed to members to show an increased interest in the fund. It was decided that an appeal should be made on the Branch agenda papers and also through the local associations.

Officers.

The following officers were elected:

Trustees: Dr. R. H. Crisp, Dr. J. J. Holland, Dr. L. E. Le Souef.
Councillor: Dr. J. L. Day.
Auditors: Dr. B. C. Cohen and Dr. T. C. Boyd.
Secretary and Treasurer: Dr. J. R. G. Maitland.

The Anatomy School.

Dr. H. J. Gray, on behalf of the subcommittee, submitted the Anatomy School report as follows:

The Anatomy School has been used throughout the year by dental students under the supervision of Dr. E. Thompson, and several members have dissected parts.

During the vacation some medical students also dissected.

The committee is of opinion that efforts should be made as opportunity offers to obtain accommodation at the University, as the present rooms are uncomfortable, unhealthy and inadequate.

The report was received and adopted.

It was resolved that the Council should be asked to endeavour again to secure accommodation for the Anatomy School at the University and, if possible, in the new science building.

Post-Graduate Committee.

The report of the Post-Graduate Committee was received and adopted. The report is as follows:

The committee finished the year with a credit balance of £1 14s. 2d. in the English, Scottish and Australian Bank. To this must be added £142 17s. 6d. in fixed deposit.

The enrolment for our last course was 48—our only source of income—so that receipts balanced expenditure. Of these 48 members, 11 were country members. The falling off in enrolment of country members was due in the majority of instances to the difficulty of obtaining *locum tenentes*.

On account of the Victorian Centenary Congress later in the year the committee has suggested that no post-graduate course be held in 1935.

Personnel.—Dr. Buttsworth has been granted one year's leave. Dr. H. Stewart was appointed by the Committee to fill the casual vacancy of surgeon due to Dr. F. J. Clark's resignation. Dr. G. A. Thompson (obstetrics) and Dr. F. J. Clark (surgeon) retire from the committee this year and their places have to be filled.

Election of Members.—Dr. J. P. Ainslie (surgeon) and Dr. S. E. Craig (gynæcologist) were appointed to the vacancies on the committee.

Votes of Thanks.

Dr. F. L. Gill referred in eulogistic terms to the services of Dr. H. J. Gray and Dr. H. B. Gill; they were accorded a vote of thanks.

On the motion of Dr. James Bentley, seconded by Dr. A. Syme Johnson, the meeting recorded its appreciation of the work done in the past by Dr. F. A. Hadley, and expressed the hope that his health would improve. It was resolved that a letter be written to Dr. Hadley.

A vote of thanks was accorded to Dr. R. H. Crisp for his untiring efforts on behalf of the Branch during the previous twelve months, and for his president's address.

The meeting closed with a vote of thanks to Dr. James Bentley and his staff for their hospitality in connexion with the annual meeting.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held on April 24, 1935, in the Prince of Wales Building, University of Adelaide, DR. F. ST. J. POOLE, the Acting President, in the chair.

Pyelitis and its Medical Treatment.

DR. A. R. SOUTHWOOD read a paper entitled: "Pyelitis and its Medical Treatment" (see page 35).

DR. I. B. JOSE stressed the need for determining the cause of pyuria, particularly when the condition had come to the chronic stage. The difficulty was at what period one should advise putting the patient to the inconvenience and expense of a urological investigation, and how long should one proceed with urinary antiseptics, ketogenic diet *et cetera* before turning the patient over to a cystoscopist for an examination, which should always be complete. One should not be satisfied with a negative result in a plain X ray examination for stone.

His plan was, if after the first acute attack the urine was not free from pus and the culture free from organisms in eight weeks, X ray examination, cystoscopy, and pyelography, if required, should be performed. If the result were negative, he proceeded with renal lavage.

A considerable number of cases were cleared up under a few treatments. The numbr of treatments required were controlled by urine culture tests. If, after a dozen weekly treatments, the infection still persisted, the whole case should be reviewed for foci of infection and the urological examination should be repeated. If the result was negative, ketogenic diet should be applied. In cases of recurrent attacks of acute pyelitis the same procedure was adopted in a quiet interval.

Dr. Jose considered that on the whole too often the case was allowed to proceed for too long a period on alkalis or antiseptics, hoping that the condition would improve, as the symptoms had generally been temporarily relieved.

Dr. Jose also referred to cases of cystitis due to cystocele which were missed and dubbed chronic pyelitis because of lack of an efficient pelvic examination. The remedy, of course, was obvious.

Dr. Jose illustrated his remarks by a number of skiagrams and pyelograms of conditions which had been discovered during routine urological examination in cases referred to as chronic pyelitis.

DR. W. J. CLOSE thanked Dr. Southwood and Dr. Jose for the very interesting matter presented. He had been particularly interested in the lucid way the medical treatment by ketogenic diet had been set out.

He was rather disappointed to find the urological examination placed so far down on the list. Dr. Jose's limit of six to eight weeks' treatment of the acute infection seemed reasonable, after which one might consider the condition becoming chronic and necessitating a more scientific investigation.

The type of organism was important. Dr. Southwood had no doubt limited his remarks to the *Bacillus coli communis* cases, which probably constituted 90% of all, but infection with the staphylococcus and other urea-splitting organisms should call for X ray examination and pyelography, as stone should be suspected. In that class of case more frequently than not nephrectomy was ultimately found to be necessary.

Even in acute cases there was one type that should be investigated straight away, and that was the hyperacute cases occurring during pregnancy. Catheterization of the ureters would give one valuable information in such cases, or at least provide all-important drainage. One such case recently was found to be due to a stone impacted at the fork of a bifid ureter. The kidney had to be explored and was found to be a typical surgical kidney necessitating nephrectomy.

Dr. Close had not yet been convinced that the apparent effect of ketones was not in reality due to the high urine acidity. In his sojourn as honorary medical officer at the Children's Hospital he had seen a number of children who had undergone medical treatment in the wards by the ketogenic method, and in some instances pelvic lavage had been given a trial. In these cases he had found the quick change-over from alkaline to extremely acid urine, combined with hexamine and a protein diet, to be usually effective.

Referring to the effect of a cystocele and the resulting chronic basal cystitis mentioned by Dr. Jose, Dr. Close was loath to stress the importance of a cystocele. Too often colpotomy had been performed for this, and numerous other gynæcological operations, when the actual focus was purely urethral. Many women came up for investigation after years of suffering, the diagnosis having been pyelitis or cystitis, recurrent or chronic, and the urethra, usually the neighbourhood of the internal meatus, had been the only site of inflammation to be found. The treatment of the urethra was very simple and by no means operative, but these women had usually undergone numerous surgical adventures. Dr. Close was unable to say whether the urethra in these cases was a starting point for repeated ascending infections or the residue of a past general urinary infection. At all events, he was of the opinion that the cystocele was seldom the cause of urinary symptoms or the origin of recurrent pyelitis.

Regarding pelvic lavage, the passage of catheters up the ureters, as Dr. Jose said, had frequently the major effect in allowing drainage, but he believed that the astringent effect of silver nitrate contributed largely.

He felt that in difficult cases ringing in the changes was important—alkalis, hexamine with ammonium chloride and ketogenesis, hexyl resorcinol, and a few of the aniline compounds, in rotation, being worth a trial. The intravenous use of antiseptics was questionable, but he had sometimes used mercurochrome 0·4% with glucose, increasing the dosage of mercurochrome from 40 to 140 milligrammes in adults, and he believed it valuable as a change-over.

DR. S. R. BURSTON stressed the necessity of thorough radiographic investigation of cases of pyelitis which did not clear up under treatment in a matter of six to eight weeks. With regard to the ketogenic diet, he considered that it was difficult for most patients to manage it during the summer months in this country. He had used it in a number of cases and found that in those in which he could get a satisfactory acidity it was successful, providing no complications were present.

With regard to Dr. Close's queries as to whether the ketonuria or the acidity of the urine was the important factor, he pointed out that Murray Lyon and others had amply demonstrated that rendering the urine acid was not effective unless, at the same time, a heavy ketonuria was induced.

DR. FRANK BEARE said that he wished to ask Dr. Southwood several questions.

First, he could not agree with Dr. Southwood when he said that rigors were of common occurrence in the pyelitis of infants. In his experience at the Babies' Hospital he had seen very few infants with rigors; in fact, off hand he could not recall any. It was a well known fact, he thought, that rigors were rare in infants and, for that matter, in young children. They seemed to be given to having convulsions on those occasions when a similar state of affairs in adults would lead to rigors. He asked for any observations that Dr. Southwood had made in that respect.

Secondly, as regards treatment, he wished to know if hexamine given in watery mixtures was stable. He instanced the late Dr. Frank Kidd, who gave it in tablet form for safety, to be sure that the drug had not broken down. Dr. Beare also wished to know whether Dr. Southwood had used any urinary antiseptic acting in alkaline media, such as the mixture containing benzoate of ammonia, bicarbonate of soda, boracic acid, saccharin, and infusion of serpentaria. He gave an account of a case of a pharmacist who was suffering from a pan-infection (with *Bacillus coli communis*) of the urinary tract (pyelitis, prostatitis, urethritis and epididymitis), who had resisted treatment till put on this mixture. He had had other similar experiences.

Finally, he wished to know how long urine could be kept after being voided when determining the hydrogen ion concentration. He thought this was a point of practical importance in the ketogenic treatment of pyelitis described by Dr. Southwood.

Dr. Southwood, in reply, thanked Dr. Jose for the excellent demonstration of X ray pictures and the reports of cases. The subject required the cooperation of physician, surgeon, pathologist and radiologist. Dr. Southwood agreed that obstinate cases should not be kept too long without a full urological investigation. Patients failing to respond well after eight weeks of thorough medical treatment, including hexamine and ketogenic diet, should certainly be sent to the urologist. Some obstructive or seriously destructive lesion would probably be found in such resistant cases. Wilson, in London, found that 88% of pyelitis cases responded well to ketogenic dieting. The diet gave results quickly, if at all. One question was whether urological investigation should be done before or after trying the ketogenic diet. Unless the surgical indication was definite, Dr. Southwood strongly advised the diet treatment as the next step if hexamine failed. Alkalis, hexamine and then ketogenic diet was the correct sequence. Wilson said that the patient should have the situation explained to him and be allowed to choose; he might be one of the large group responsive to diet. Ketogenic diet was unpleasant, and some patients might find a vigorous urological investigation not altogether to their liking.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Royal Melbourne Hospital on May 15, 1935. The meeting took the form of a series of demonstrations by members of the honorary staff. Parts of the report of this meeting were published in the issues of June 8 and 22 and July 6, 1935.

Polycystic Disease of the Kidneys.

DR. W. W. S. JOHNSTON showed three patients to illustrate various phases of polycystic disease of the kidneys. Amongst the features of interest displayed by this series were the familial nature of the condition and the mildness of symptoms occurring in some cases, despite gross changes in the kidney substance. In two of the patients shown, each being over seventy years of age,

there was no obvious deterioration in general health, notwithstanding the existence of the condition in a well established form.

The first patient was a married woman, aged seventy-one years, who had applied for treatment at the hospital on April 2, 1935, on account of weakness in the legs. The heart was somewhat enlarged and the blood pressure was raised. The systolic pressure was 210 and the diastolic 100 millimetres of mercury. The urine was found to be free of albumin and of abnormal cells and casts. Abdominal examination revealed the presence of a large irregular mass, nodular in character, in the region of the kidney on each side. The maximum figure in the urea concentration test was 1·6% in the second hour. An X ray examination of the urinary tract had shown gross enlargement of both kidneys, with irregular, apparently cystic, outline strongly suggestive of polycystic disease. Subsequently Dr. J. T. Tait, by pyelography, had shown bilateral polycystic kidneys, the left having undergone ptosis with clubbing of the calyces and the right being less irregular.

The second patient, aged seventy-five years, married, was a sister of the previous patient and had reported at the hospital on March 14, 1935, because of intermittent attacks of diarrhœa and vomiting. At that time, on routine examination nothing abnormal was found, and after the subsidence of her symptoms she had ceased to attend. She was reexamined on April 2, when she had accompanied her sister to hospital, and similar but less definite signs were discovered on abdominal palpation. A few granular casts and occasional pus cells were seen in the urine, which was free of albumin. The systolic blood pressure was 156 and the diastolic pressure was 90 millimetres of mercury. The urea concentration test yielded a maximum figure of 2·1% at the end of the second hour. X ray examination of the urinary tract revealed some enlargement of both kidney shadows, and a pyelogram taken by Dr. Tait showed bilateral polycystic changes more definite on the left side. There was blunting of the calyces on each side.

Dr. Johnston's third patient was a married woman, aged thirty-five years, who had been in the hospital from September 4, 1927, to October 4, 1927, with symptoms of uræmia and hypertension. The systolic blood pressure reading had been 210 and the diastolic 160 millimetres of mercury. She had had albuminuria of pregnancy five years earlier and persistent albuminuria had followed a second pregnancy five months before her admission to hospital. During her subsequent attendance at the out-patient department her history was typical of chronic nephritis with hypertension. The urine had shown at most only a trace of albumin. On April 24, 1931, the blood urea amounted to 35 milligrammes per 100 cubic centimetres, and on June 22, 1934, to 30 milligrammes. The highest concentration reached in a number of urea concentration tests was 2%. On July 19, 1934, the maximum concentration was 1·75% at the end of the second hour.

On June 19, 1934, she complained of vomiting after meals for the previous few weeks, and on examination a large irregular mass was found in each loin. Radiographically the kidney shadows were indistinct, but appeared to be larger and rather more lobular than normal. Pyelography had not been undertaken in view of the possible ill-effects on the already advanced condition of chronic nephritis. As the diagnosis of polycystic disease appeared to be established at this stage, it was decided to obtain further details of the final illness of the patient's mother, who was said to have died in the Melbourne Hospital in 1905 from Bright's disease. Reference to the notes, including an autopsy report, showed that she had died from polycystic disease of the kidneys, liver and thyreoid. Although several relatives of the present patient, including a maternal aunt and two sisters, had been examined subsequently, nothing to indicate the presence of polycystic disease of the kidney had been found in these people.

Again recently the patient had been admitted to hospital with symptoms of uræmia, but had improved sufficiently to be transferred to the out-patient department.

Spinal Tumour.

DR. G. A. PENINGTON presented a male patient, aged fifty-one years, upon whom Dr. B. T. Zwar had operated for a tumour of the spine.

In October, 1933, the patient attended at the hospital, complaining of weakness in the legs for one year and unsteadiness of gait. The right leg was affected to a greater extent than the left with loss of sensation in the right foot. He had experienced cramp-like pain in the abdomen at times and, for nine months, there was a tendency to fall on coughing or on sneezing. Four months before he had had severe pain of short duration in the left scapular region. He had an attack of influenza in 1919, and in 1926 he acquired syphilis. Apart from pyorrhœa, the only abnormal findings were neurological: nystagmus to the left, decreased power and spasticity of the right lower limb, gross impairment of pain appreciation, of thermal discrimination, of joint and muscle sense and slight impairment of tactile sensation in both lower limbs up to the groin, the right leg being affected to the greater degree. The gold sol test gave a luetic curve, but no reaction was obtained to the Wassermann test both in blood and cerebro-spinal fluid. The globulin content of the cerebro-spinal fluid was increased. There was a quick rise and fall in the Quickenstedt test. Flocculation tests gave no reaction after a provocative injection of "Novarsenobillon". On radiographic examination it was noted that there was slight lipping and slight wedging of the eleventh dorsal vertebral body. It was considered that the patient had disseminated sclerosis and he was transferred to the out-patient department.

He was readmitted to hospital on May 30, 1934, because pain had recurred in the lower part of the abdomen two months previously and developed in the right leg with sudden flexor spasms affecting the right leg chiefly. Weakness of the left leg became apparent and was progressing, and though his bladder control was good, occasionally he experienced poor rectal control. There was demonstrable impairment of the posterior and lateral columns of both sides below the level of the twelfth thoracic vertebra, but the right side was more greatly affected. Apart from slight increase in globulin and the presence of only two cells per cubic millimetre, investigation of the cerebro-spinal fluid revealed no abnormality; this investigation included normal findings for the colloidal gold curve and Quickenstedt test. On cisternal puncture and introduction of descending lipiodol there had been an arrest at the distal border of the ninth dorsal vertebra. Laminectomy was advised but refused by the patient.

He returned and was readmitted on April 22, 1935. For five months the weakness had increased and he had become unable to walk and had a dull aching pain on the right side of the abdomen. The neurological findings of interest were those of increased weakness in the left lower limb with patellar and ankle clonus on both sides, impaired localization of light touch below the level of the tenth thoracic area on both sides, poor discrimination of two points, impaired vibration sense over the left tibia with G 48, though no impairment on the right tibia and no impairment on either side was apparent with C 256. There was gross impairment of painful and thermal sensation and less impairment of tactile sensation below the tenth thoracic level. A band of hyperalgesia was present at the tenth thoracic level after lumbar puncture. The patient had irregular sphincteric control with retention of urine. Lipiodol *descendens* was still held up and lipiodol *ascendens* was held at the ninth thoracic vertebra.

On May 7, 1935, Dr. B. T. Zwar operated under intratracheally administered anæsthesia with the patient in the prone position. He made a vertical incision 15·0 centimetres (six inches) long, centred opposite the ninth thoracic spinous process. Laminectomy of the seventh to tenth processes was performed and a small trephine opening was made in the lower end of the incision, and when it was extended a tumour could be felt and seen through the *dura mater* and seen through the dura. The dura and the arachnoid membranes were adherent to each other and to the tumour. The tumour was firm and well encapsuled and, by sharp dissection, was removed complete

and the dura was closed. Two needles of radium were placed one centimetre apart over the region of the tumour.

On section, the tumour was identified as a fibroid type of meningioma. A week after operation, there was practically no change in the motor signs, but, though impaired, sensation was returning, crude sensibility recovering first.

Tabes Dorsalis with Wasting of the Small Muscles of the Hands.

Dr. Penington also showed two patients with wasting of the small muscles of the hands. The first had the classical signs of *tabes dorsalis*; the other did not show any of the sensory changes, but had typical Argyll-Robertson pupils.

The first patient was a man, aged sixty-eight years, who had attended the hospital in 1920, complaining of failing eye-sight and difficulty in holding his urine. It was found that the blood yielded a strongly positive Wassermann reaction and that some wasting of the right thenar eminence had occurred. Gradually he became enfeebled and by 1925 extensive wasting of all the small muscles of the hands was demonstrable, always worse on the right side, and he had been troubled by severe tabetic pains of various types. Though the anti-specific treatment had been irregular and incomplete, his blood had ceased to react to the Wassermann test.

The second patient, a man of sixty-four years, had hemiparesis on the left side in August, 1934, from which he made a complete recovery. The blood and cerebro-spinal fluid failed to yield the Wassermann reaction. In addition to the Argyll-Robertson pupils he manifested wasting of the small muscles of both hands, especially on the right hand, which had been noticed for seven years and had been steadily progressive.

Disseminated Sclerosis with Partial Brown-Séquard Lesion.

Dr. Penington's last patient was a male, aged nineteen years, who had been admitted to the hospital on February 7, 1935, complaining of numbness of the right leg, pain on the left side of the chest for two months and dragging of the left leg for one month. For the first month numbness had extended to the right groin, but during the month before admission to hospital had moved upwards to the level of the nipple. Nothing abnormal was found except on examination of the nervous system. The right pupil was larger than the left with hippus. No weakness was detected about the trunk or the upper limbs, but there was loss of power and some spasticity of the left lower limb, with left-sided knee and ankle clonus and Babinski reflex. There was dissociation of sensation; vibration sense, localization, discrimination and joint muscle senses were normal; pin-prick sensation on the right side was grossly impaired below the seventh thoracic level, and diminished over the sixth and seventh segmental areas, and on the left side there was a zone of hyperæsthesia from the fourth to the seventh thoracic levels; temperature sensibility was lost below the seventh thoracic level on the right side and was hyperæsthetic on the left side in the same zone as the tactile sensibility. Tenderness was elicited on percussing the fourth to the seventh thoracic spines. Lipiodol *descendens* fell to the sacrum. All the special investigations—Wassermann test, lumbar puncture, cerebro-spinal fluid examination, Quickenstedt test, hydatid complement fixation test, examination of visual fields and fundi—failed to reveal any abnormal responses.

On February 17, 1935, a fine nystagmus had been noted, especially on looking to the right. By February 20, 1935, the sensory loss extended only to the ninth thoracic level. On February 25, 1935, a zone of hyperæsthesia appeared on the inner aspect of the left arm, over the second and third thoracic segments and loss of power followed in the left upper limb. On March 7, 1935, the patient had throbbing pain and weakness in the left arm and leg and there was impaired vibration sense and poor appreciation of light touch over the right leg. Pain and temperature were not appreciated below the tenth segment on the right side. On March 14, 1935, the patient complained of mistiness of vision, but the fields of vision and the fundi were not abnormal. On May 2, 1935, he lost all feeling in the third, fourth and fifth digits of the right hand, which gradually extended to the other digits. By May 9, 1935, there was some improvement, but the left leg was still spastic and there was decreased sensation to pin-prick on the right side to the sixth thoracic level.

Hydatid Cysts of the Spinal Canal.

DR. T. J. F. FRANK presented a man, aged thirty-two years, who was admitted to hospital on March 6, 1935. Three months earlier a pain had developed in the middle of the spine which, several weeks later, had radiated upwards and downwards. A month after the onset of symptoms the patient noticed an aching pain in the lower part of the chest anteriorly, and, three weeks before admission, his legs became weak and his body numb up to the level of the umbilicus. There was no disturbance of micturition or defæcation. On examination the abnormal findings were neurological: slight spastic paraplegia, active knee jerks, ankle clonus and bilateral Babinski reflex. There was no gross sensory disturbance, but at the level of the sixth thoracic segment there was a band of hyperæsthesia. The cerebro-spinal fluid examination on March 6, 1935, revealed a slight increase in globulin content, thirty-five lymphocytes and one large mononuclear leucocyte per cubic millimetre. The blood and cerebro-spinal fluid did not give the Wassermann reaction. On March 12, 1935, the optic disks were normal in appearance. On April 2, 1935, descending lipiodol was blocked at the level of the second thoracic vertebra and the lower edge was bifurcated slightly. These findings were suggestive of the presence of a spinal tumour. Three days later the Casoni skin test gave a positive reaction. Radiological examination of the chest revealed no abnormality. On April 9, 1935, ascending lipiodol was arrested at the level of the twelfth thoracic vertebra and had not moved upwards after twenty-four hours. The cerebro-spinal fluid failed to yield the hydatid complement fixation reaction; but on April 7, 1935, the blood gave a single positive in the warm method and a triple positive reaction in the icebox technique. The pre-operative diagnosis of hydatid disease of the spinal cord was made on this evidence.

On April 12, 1935, Dr. Hailes performed laminectomy from the fourth cervical to the fifth thoracic spinous processes. The dura was opened and at once hydatid membrane and daughter cysts bulged through the opening. It seemed probable that the hydatid infestation arose anteriorly from the lower cervical vertebræ, but this had not been proved. By means of a sucker, hydatid cysts were removed from as low down as the level of the first lumbar vertebra.

Since the operation the patient felt well, but had experienced pains across the chest and signs could still be elicited indicative of involvement of the lateral columns.

Dr. Frank stated that though these cysts were apparently secondary to hydatid disease elsewhere in the body, the source had not been discovered. Many cases of hydatid cysts of the vertebræ had been recorded and occasionally they invaded the spinal canal and caused pressure on the cord. Other cysts, arising in the loose fatty tissue in the spinal canal, had been recognized. With growth, the dura became thinned and penetration with rupture into the subdural space would follow. The prognosis as regards complete cure Dr. Frank regarded as hopeless, since it was impossible to remove all of the cysts. Laminectomy had relieved the compression symptoms and the same procedure could be repeated again if necessary.

Charcot's Joints.

Dr. Frank's second patient was a man, aged sixty years, with gross tabetic arthropathy of the hypertrophic type affecting both knee joints. During the twenty years in which the patient had been attending the out-patient department of the hospital with *tabes dorsalis*, the right knee had been increasing in size and it had been recognized as an example of Charcot's arthropathy for many years.

For the past eight years there had been swelling and pain in the left knee joint. Both knee joints showed gross periarticular swelling, irregular bony outgrowths, with limitation of movement and considerable grating unaccompanied by pain. There had been increasing deformity with *genu varum*. Recent skiagrams were exhibited.

Dr. Frank referred to the tendency of this affection to attack the knee and ankle joints and to the starting pains that might disturb sleep. The excessive destruction of the articular surfaces with synovial effusion and growth of osteophytic outgrowths around the margins of the articulating surfaces were readily apparent in this case.

Pernicious Anæmia and Subacute Combined Degeneration of the Cord.

Dr. Frank also showed a woman, aged thirty-four years, who was admitted to the hospital on March 25, 1934, after five years of ill-health. For that period she had been noticeably pale and was becoming progressively weaker in the body. For two years this weakness had affected her legs until she was not able to walk. The lower extremities had become numb and tingling had been present in the finger tips. The other symptoms were sore tongue, dizziness, dyspnœa on exertion, and swelling of the feet. The abnormal neurological findings were: considerable loss of power in the lower limbs, very active knee and ankle jerks, extensor plantar reflexes, loss of vibration sense, impairment of light touch and tactile discrimination over both lower limbs. On the basis of these findings a diagnosis of subacute combined degeneration of the cord was made.

On March 27, 1934, a complete blood examination had been made and a test meal had shown achlorhydria with total acidity less than ten. The results of the initial blood examination and of subsequent examinations are shown in the accompanying table. The patient's nervous degeneration had been associated with pernicious anæmia. On March 28, 1934, the blood failed to give the Wassermann reaction. There had been a delayed positive Van den Bergh reaction, with the presence of four units of bilirubin. The fundi and optic disks were normal in appearance.

The treatment consisted of a combination of two ounces of "Suso-ventrin" daily by mouth, with depôt injections of liver extract intramuscularly. The dose of eight cubic centimetres was given twice a week for two weeks, and then ten cubic centimetres were injected once a month to date. Iron and ammonium citrate in doses of 1·0 gramme (fifteen grains), at first three times and latterly twice a day, had been used as well.

Dr. Frank said that the result of treatment had been very satisfactory and that the treatment was simple to carry out. It was relatively inexpensive and the depôt injections helped enormously to make up for uncertainty as to the faithfulness of the patients who could not be depended upon to take liver preparations by mouth. Large doses of a suitable iron preparation were indicated specially in subacute combined degeneration of the cord, and when this addition was made to the treatment patients had progressed who, on liver treatment alone, were not improving.

ANNUAL MEETING, MELBOURNE, 1935.

CERTAIN alterations and additions to the programme already published in this journal are announced. Members are asked to make careful note of each of the items.

Monday, September 9.

At 4 p.m. there will be a civic reception of the official party, of delegates and certain prominent visitors.

In place of the reception by the Victorian Medical Women's Society announced for 8.30 p.m., a dinner will be given at 7 p.m. by the Victorian Medical Women's Society to all interstate and overseas medical women.

At 9 p.m. a reception will be held by the State Government of Victoria in the National Gallery.

Tuesday, September 10.

The President's reception at 8.30 p.m. will be followed by a dance.

Wednesday, September 11.

The garden party to be held at the University of Melbourne will be preceded by a gathering in the Wilson Hall, at which honorary degrees will be conferred by the University of Melbourne.

Friday, September 13.

At 9 a.m. play will begin for the Leinster and Childe Golf Cups for men at the Metropolitan Club.

At 9.30 a.m. play will commence for the Notts Ladies' Challenge Golf Cup at the Peninsula Club, Frankston.

At 8 p.m. a popular lecture will be given by the Right Honourable Lord Horder, K.C.V.O., D.C.L., M.D., F.R.C.P.

Saturday, September 14.

Many entertainments and excursions are being arranged.

Clinical Addresses.

The following addresses will be delivered during the week at 8.45 a.m.:

"Specialism: Neurology and Medicine", by Professor Edwin Bramwell, M.D., F.R.C.P. (Edinburgh).

"The Valley of Dry Bones", by Professor E. W. Hey Groves, M.D., M.S., F.R.C.S.

"Œsophageal Obstruction", by H. S. Souttar, M.D., F.R.C.S.

"The General Practitioner and Forceps", by Sir Ewen Maclean, T.D., F.R.C.P., F.C.O.G.

"The Dyspepsias of Childhood", by Robert Hutchison, LL.D., M.D., F.R.C.P.

Date.	Hæmoglobin Value (Sahli), *Per Centum*.	Red Blood Cells, per Cubic Millimetre.	Leucocytes, per Cubic Millimetre.	Colour Index.	Remarks Concerning Blood Films.
March 27, 1934 ..	53	2,440,000	5,300	1·10	Anisocytosis. Large macrocytes and misshapen microcytes. No polychromasia. Relative lymphocytosis. Reticulocytes under 1%
April 4, 1934	58	2,830,000	5,750	1·03	As before
April 14, 1934 ...	65	3,770,000	9,300	0·87	As before
April 19, 1934 ...	72	4,070,000	8,000	0·90	Still some macrocytes
August 27, 1934..	84	5,000,000	14,100	0·84	No abnormal forms
January 10, 1935 .	94	4,620,000	13,200	0·81	No abnormal forms
May 9, 1935	80	4,290,000	9,900	0·85	No abnormal forms

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Rolph, William Henry, M.B., B.S., 1935 (Univ. Melbourne), 3, Blyth Street, Brunswick, N.10.

Eddey, Howard Hadfield, M.B., B.S., 1934 (Univ. Melbourne), Melbourne Hospital, Melbourne, C.1.

MEMORIAL TO THE LATE SIR RICHARD STAWELL.

To perpetuate the memory of the late Richard Rawdon Stawell, it has been decided to establish a fund, from the income of which will be provided a Triennial Prize to be awarded for an essay on some medical subject of clinical significance.

The award will be made coincident with each Australasian Medical Congress, and will be open to all graduates of Australian universities of not more than three years' standing.

The fund will be administered by trustees appointed by the Council of the Victorian Branch of the British Medical Association.

The Honorary Treasurer of the fund will be Dr. C. H. Mollison, 41, Spring Street, Melbourne, C.1, and subscriptions are invited from all Australian members of the British Medical Association, from any institutions, societies or clubs with which the late Richard Rawdon Stawell was associated, and from any personal friends who may wish to contribute.

Post-Graduate Work.

WEEK-END POST-GRADUATE COURSE AT LISMORE.

THE New South Wales Permanent Post-Graduate Committee, in conjunction with the North-Eastern Medical Association, will hold a week-end course at Lismore on Saturday, August 10, and Sunday, August 11, 1935. The programme is as follows:

Saturday, August 10.

2 p.m.—General meeting of the North-Eastern Medical Association at the Commercial Hotel, Woodlark Street, Lismore.

4 p.m.—Afternoon tea and registration for the course.

4.15 p.m.—Lecture: "Modern Diagnosis and Treatment of Acute Bronchitis, Pleurisy and Pneumonia", Dr. A. J. Collins.

7 p.m.—Dinner.

8 p.m.—Symposium, "Chronic Rheumatism": medical aspects, Dr. A. J. Collins; orthopædic aspects, Dr. D. J. Glissan.

Sunday, August 11.

(At the Lismore Base Hospital.)

9 a.m.—Demonstration of plaster of Paris technique, Dr. D. J. Glissan.

2 p.m.—Medical cases, demonstration by Dr. A. J. Collins.

4 p.m.—Manipulative surgery, lecture-demonstration by Dr. D. J. Glissan.

7.30 p.m.—Lecture: "Diet in Diabetes and Disease Generally", Dr. A. J. Collins.

The fee for the course will be one guinea. Those intending to be present at the course are requested to notify Dr. J. R. Ryan, Lismore, as soon as possible, and to state whether it is their intention to be present at the dinner.

Correspondence.

PRICES OF PROPRIETARY REMEDIES.

SIR: Of recent years chemists throughout New South Wales have been considerably inconvenienced due to the habit of some members of the medical profession quoting prices of proprietary medicines to their patients.

It would probably matter little if they quoted the approximate retail prices, but unfortunately this is not done, more often than not for the simple reason that they do not know them.

What is often done is that the patient, on inquiring how much such and such a prescribed remedy will cost, is informed of the wholesale price of the particular line to the profession and the chemist. The result is that when the patient obtains the line from the chemist and is charged a higher price, he or she feels that the chemist is overcharging.

It will, of course, be fully realized by the profession that a chemist must add something to the wholesale price of a line to cover his overhead expenses (no small item) and to make his profit, and this something has been definitely laid down for him by his organization, the Guild. He is thus prevented from overcharging, and complaints in this regard would be quickly investigated by the Guild.

Manufacturers of ethical proprietary lines have been approached with the object of having a note inserted in their price lists to the effect that the prices quoted are to the profession only and that the goods will be supplied by any chemist at the retail price set forth in current Guild lists. Some have already adopted this idea, but until such a method becomes universal pharmacists must look to the medical profession for assistance. Just as a practitioner would resent the discussion of his fees by a chemist with his customer, so is a chemist entitled to be upset at a discussion of prices by a practitioner with his patients.

There are, it is realized, times when a patient is in indigent circumstances and unable to afford the price of some of the more expensive lines, and in such cases, were a practitioner to indicate such a fact in some way on the prescription, there are few chemists who would not meet the case.

Pharmacists generally are only too pleased to cooperate with the medical profession in the interests of public health, and any reciprocal cooperation of the nature above referred to will be very greatly appreciated.

Yours, etc.,

REG. E. GOSTELOW,
Secretary,
Federated Pharmaceutical Service Guild of Aust. (N.S.W. Branch).

June 21, 1935.

Obituary.

GEORGE HERBERT BENNETT.

WE regret to announce the death of Dr. George Herbert Bennett, which occurred on June 28, 1935, at Gerringong, New South Wales.

LAURENCE LINDLEY POLLOCK PATERSON.

WE regret to announce the death of Dr. Laurence Lindley Pollock Paterson, which occurred on July 5, 1935, at McLeod, Victoria.

SAMUEL HENRY HUGHES.

WE regret to announce the death of Dr. Samuel Henry Hughes, which occurred on July 7, 1935, at Killara, New South Wales.

NOTICE.

SYDNEY UNIVERSITY GRADUATES' ASSOCIATION.

ON Wednesday, July 24, 1935, at 8 p.m., a meeting of graduates of the University of Sydney will be held in the Great Hall at the University for the purpose of founding a Sydney University Graduates' Association which, it is hoped, will function in a manner similar to graduates' associations in other universities. It will endeavour to promote corporate action by graduates in matters affecting their interests and keep them in touch with one another after their university life is ended. In order to achieve these objects it intends to seek the cooperation of the various faculty graduates' societies already in existence.

A provisional committee has been formed to carry out the necessary work to effect the formation, and of this committee Mr. P. C. Spender, K.C., is chairman, and Mr. P. J. Kenny, B.A., Secretary.

Books Received.

INDIVIDUAL HEALTH: A TECHNIQUE FOR THE STUDY OF INDIVIDUAL CONSTITUTION AND ITS APPLICATION TO HEALTH, by E. Obermer; Volume I: Biochemical Technique, by E. Obermer and R. Milton; 1935. London: Chapman and Hall, Limited. Demy 8vo., pp. 260, with illustrations. Price: 15s. net.

PHYSICAL CHEMISTRY FOR STUDENTS OF BIOLOGY AND MEDICINE, by D. I. Hitchcock, Ph.D.; Second Edition; 1934. Springfield: Charles C. Thomas; London: Baillière, Tindall and Cox. Royal 8vo., pp. 225, with illustrations. Price: 12s. 6d. net.

THE TREATMENT OF RHEUMATISM IN GENERAL PRACTICE, by W. S. C. Copeman, M.A., M.B., B.Ch., M.R.C.P., with foreword by W. Hale-White, K.B.E., M.D., F.R.C.P., Hon. LL.D.; Second Edition; 1935. Demy 8vo., pp. 236. Price: 9s. net.

Diary for the Month.

JULY 16.—Tasmanian Branch, B.M.A.: Council.
JULY 16.—New South Wales Branch, B.M.A.: Ethics Committee.
JULY 17.—Western Australian Branch, B.M.A.: Branch.
JULY 17.—Victorian Branch, B.M.A.: Clinical meeting.
JULY 18.—New South Wales Branch, B.M.A.: Clinical meeting.
JULY 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JULY 24.—Victorian Branch, B.M.A.: Council.
JULY 25.—South Australian Branch, B.M.A.: Branch.
JULY 25.—New South Wales Branch, B.M.A.: Branch.
JULY 26.—Queensland Branch, B.M.A.: Council.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," pages xvi, xvii.

AUSTIN HOSPITAL FOR CANCER AND CHRONIC DISEASES, HEIDELBERG, VICTORIA: Honorary Ear, Nose and Throat Surgeon.

BUNDABERG HOSPITAL, BUNDABERG, QUEENSLAND: Assistant Medical Officer.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer.

PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Resident Medical Officers.

PRINCE HENRY HOSPITAL, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officers.

PUBLIC SERVICE BOARD, ADELAIDE, SOUTH AUSTRALIA: Resident Medical Officers.

THE BROKEN HILL AND DISTRICT HOSPITAL, BROKEN HILL, NEW SOUTH WALES: Resident Medical Officer.

THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Honorary Clinical Assistants, Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Chiliagoe Hospital. Members accepting LODGE appointment and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £1 for Australia and £2 5s. abroad *per annum* payable in advance.